CORRELATION OF QRS AMPLITUDE AND RECIPROCAL CHANGES IN ECG TO OUTCOME IN FIRST TIME ST ELEVATION MYOCARDIAL INFARCTION

Dissertation submitted to

THE TAMIL NADU DR.M.G.R MEDICAL UNIVERSITY

In fulfilment of the regulations for the award of the degree

M.D. GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE
PSG INSTITUTE OF MEDICAL SCIENCES & RESEARCH
THE TAMIL NADU DR.M.G.R MEDICAL UNIVERSITY
CHENNAI, TAMIL NADU

APRIL 2015

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DECLARATION

I hereby declare that this dissertation entitled **CORRELATION OF QRS AMPLITUDE AND RECIPROCAL CHANGES IN ECG TO OUTCOME IN FIRST TIME ST ELEVATION MYOCARDIAL INFARCTION** was prepared by me under the direct guidance and supervision of my Professor **Dr. L S SOMASUNDARAM,** PSG Institute of Medical Sciences & Research, Coimbatore.

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Dr.Silpita Katragadda

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February 12, 2014

To
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The Institutional Human Ethics Committee, PSG IMS & R, Coimbatore -4, has reviewed your proposal on January 9, 2014 in its expedited review meeting held at IHEC Secretariat, PSG IMS&R, between 10.00 am and 11.00 am, and discussed your study proposal entitled:

"Correlation of QRS amplitude & reciprocal changes in ECG to outcome in first time ST - elevation myocardial infarction"

The following documents were received for review:

1. Duly filled application form
2. Proposal
3. Informed Consent Forms
4. Data collection tool
5. CV
6. Budget

After due consideration, the Committee has decided to approve the study.

The members who attended the meeting at which your study proposal was discussed are as follows:

Name	Qualification	Responsibility in IHEC	Gender	Affiliation to the Institution Yes/No	Present at the meeting Yes/No
Dr P Sathyan	DO, DNB	Clinician, Chairperson	Male	No	Yes
Dr S Bhuvaneshwari	M.D	Clinical Pharmacologist Member - Secretary	Female	Yes	Yes
Dr Sudha Ramalingam	M.D	Epidemiologist Alt. Member - Secretary	Female	Yes	Yes
Dr Y S Sivan	Ph D	Member –Social Scientist	Male	Yes	Yes
Dr D Vijaya	Ph D	Member – Basic Scientist	Female	Yes	Yes

The approval is valid for one year.

We request you to intimate the date of initiation of the study to IHEC, PSG IMS&R and also, after completion of the project, please submit completion report to IHEC.

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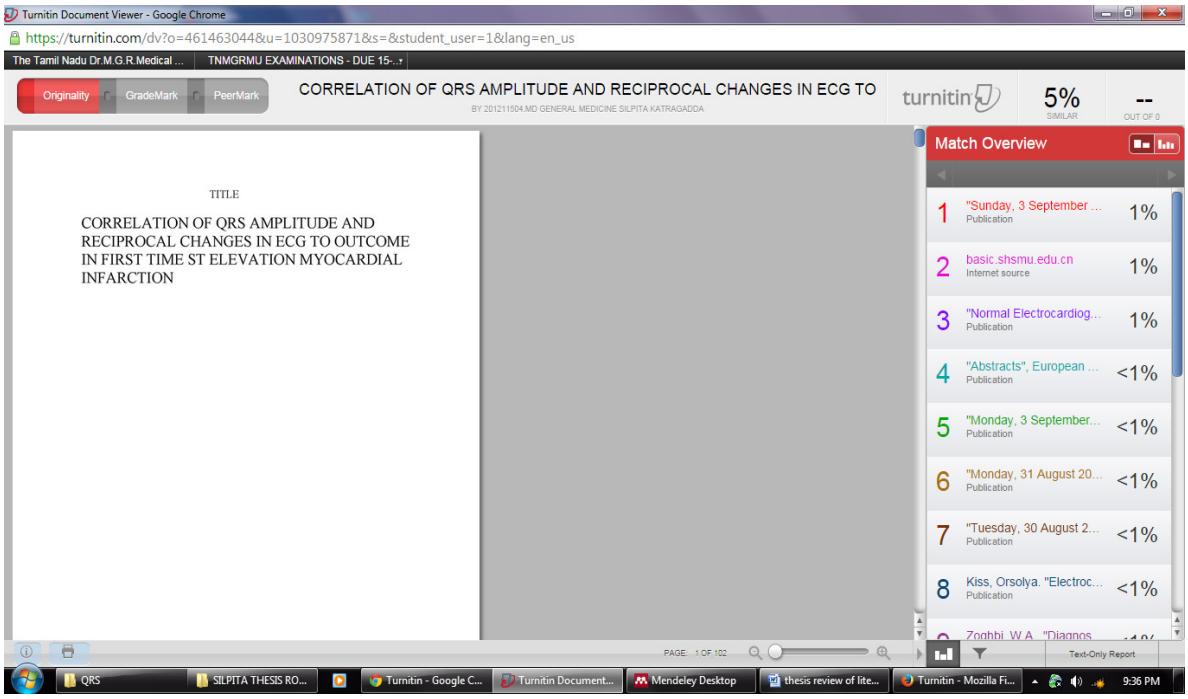
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CONTENTS

ABSTRACT

Objective: To observe the association of ECG changes with Left Ventricular function and clinical outcome in patients with first time ST elevation myocardial infarction.

Methods: One hundred and fourteen patients of acute ST elevation myocardial infarction (STEMI) (78 anterior, 36 inferior) aged between 18-90 years were studied with regard to the QRS amplitude and reciprocal lead changes in the ECG at admission which were correlated with the left ventricular dysfunction and wall motion score of seventeen segments of the left ventricle.

Results: In both anterior and inferior wall MI patients the association between presence of reciprocal changes with left ventricular dysfunction and wall motion score was found to be insignificant (p value = > 0.05).

In both anterior and inferior wall MI patients, the association between QRS amplitude score and left ventricular dysfunction was analyzed and was found to be statistically insignificant (p value >0.05).

The mean QRS amplitude score in anterior wall infarction patients was 34.17 ± 16.9. The mean Wall motion score in anterior wall infarction patients was 24.36 ± 3.9. Using the pearson chi square test, the correlation was found to be statistically significant (r value = **- 0.147**)

The mean QRS amplitude score in inferior wall infarction patients was 35.03 ± 14.4. The mean Wall motion score in anterior wall infarction patients was 21.69 ± 4.7. Using the pearson chi square test, the correlation was found to be statistically significant (r value = **- 0.359**)

Out of the 78 patients with anterior wall infarction, 35 patients had reciprocal changes in their ECGs. Among those 35 patients, 15 (42.9%)

had no symptoms NYHA class I, 14 (40%) patients had NYHA class II symptoms, 4 (11.4%) patients had NYHA class III symptoms and 2(5.7%) were lost to follow up. The p value calculated using Pearson Chi square test and was found to be statistically significant($p = 0.001$).

Out of the 36 patients with inferior wall infarction, 20 patients had reciprocal changes in their ECGs. Among those 20 patients, 9 (45%) had no symptoms NYHA class I, 10 (50%) patients had NYHA class II symptoms and 1 (5%) patient had NYHA class III symptoms. The p value calculated using Pearson Chi square test was statistically significant($p = 0.004$).

In both anterior and inferior wall MI patients, the association between QRS amplitude score and clinical outcome (NYHA class) was analyzed and was found to be statistically insignificant (p value >0.05).

Conclusions: The presence of reciprocal changes was not associated with a higher wall motion score indicating the degree of infarction.

The finding of low QRS amplitude in the ECG didn't directly lead to the development of left ventricular dysfunction. LVD was present irrespective of the QRS amplitude score.

In patients with lower QRS amplitudes, higher wall motion scores were observed indicating that low voltage of QRS complexes in the ECG can be predictive of larger extent of the infarct.

The presence of reciprocal changes in the ECG can signify poorer outcome on follow up at three to four weeks after infarction.

Low QRS amplitude is not predictive of the clinical outcome following STEMI.

INTRODUCTION

Myocardial infarction is one of the most frequently encountered reason for hospital admission and is commonly seen in all populations worldwide. Theoretically speaking, infarction of the myocardial muscle can be detected based on certain pathological features like coagulation necrosis causing loss of muscle cells (myocytes). When such a pathological change occurs, it triggers inflammation which subsequently leads to fibrosis and healing with a scar.

The clinical approach to a patient suspected to have an acute coronary syndrome consists of detailed history taking, electrocardiogram (ECG) changes, cardiac biochemistry evidence and imaging. The reliability of each of these modalities as a diagnostic tool is dependent on multiple factors, the most significant being the window period between the time of infarction and the time the patient seeks medical attention.

The current approach to patients presenting with typical or atypical features of myocardial ischemia or infarction starts with making the provisional diagnosis of acute coronary syndrome. Then, depending on the changes seen on the twelve lead ECG, acute coronary syndrome is classified into ST elevation myocardial infarction (STEMI) or non ST elevation myocardial infarction (NSTEMI).

Inspite of major leaps in the diagnostic tools and treatment of myocardial infarction, ST segment elevation MI persists as a leading cause for ill health in both the developed and developing world. The prevalence of coronary disease and infarction is on rise in the developing countries, with further worsening of adverse cardiovascular events due to disadvantages like inadequate primary prevention policies and limited availability of medical help.

The meteorical advances in acute coronary care and resuscitation since the twentieth century has led to considerable decline in mortality and morbidity rates from STEMI. In the early parts of the twentieth century, therapy was centered around passive observation and monitoring than active intervention. Significant advances have opened the gates to the current reperfusion therapy, which along with intensive hemodynamic monitoring has improved the standards of acute coronary care and emergency management. The approach to ST elevation MI is increasingly leaning towards practice guidelines and evidence based medicine.

There has been extensive research done in the field of cardiovascular medicine focused on ST elevation myocardial infarction and the dynamics of complications surrounding it. One particular area of interest in recent studies appears to be the study of extent of infarct over imaging modalities and its correlation with various events in the course of

infarction. Other parameters gaining importance are non traditional ECG changes like reciprocal changes, QRS sloping etc.

The simplest and oldest diagnostic tool for myocardial infarction is the 12 lead electrocardiograph. In daily practice, even though the ECG remains an important test in diagnosis and detection of progression of disease, there still remains ample potential for its role as a prognostic marker. The fundamental advantage that stands to be gained by realizing this potential is the time it saves for immediate intervention. In addition, it can serve as an instant and cost effective method for risk assessment instead of waiting for biochemical and angiography results. The purpose of this study is to explore the possibility of ECG being a prognostic marker in terms of left ventricular outcome.

AIM

To observe the association of ECG changes with Left Ventricular function and clinical outcome in patients with first time ST elevation myocardial infarction.

MATERIALS AND METHODS

Type of Study : Prospective study

Place of Study : PSG hospitals, PSG IMS & R , Coimbatore

Study Population : Patients hospitalized at PSG HOSPITAL after being diagnosed with ST elevation Myocardial infarction based on ST elevation seen on admission ECG.

Duration of the study : 9 months (January to September 2014)

Inclusion criteria :

- patients aged above 18 years with first time ST elevation myocardial infarction.

Exclusion criteria :

- Bundle branch blocks
- Pre-existing cardiac illness
- Moribund cases

METHODS

Patients hospitalized at PSG Hospitals, Coimbatore with the presence of ST elevation myocardial elevation on electrocardiogram between January 2014 to September 2014, meeting the specified

inclusion criteria were included in the study. Informed consent was obtained and history was taken from the patients.

The patient's age, gender, risk factors for myocardial infarction were documented at admission. The first electrocardiogram (ECG) taken was studied for presence or absence of reciprocal changes. The R wave amplitude in mm (from upper limit of the baseline to the top of the positive deflection) was taken in all the leads of the ECG and the sum was noted as the QRS amplitude score.

The type of management done for the patient, whether medical or interventional was documented. Details of echocardiogram done at 48-72 hours after infarction – left ventricular function and regional wall motion abnormality were noted. Left ventricular function was assessed based on ejection fraction using eyeballing method. Patients were graded as having mild Left ventricular dysfunction (LVD) if the ejection fraction was 45-50 %, moderate LVD if ejection fraction was 35 – 45% and severe LVD if ejection fraction was less than 35%. Regional wall motion abnormality was assessed using American society of echocardiography (ASE) method by grading wall motion of all seventeen left ventricular segments. This was represented as Wall motion score. For each of the individual 17 segments, score of 1 was taken as normal regional wall motion, score of 2 was given for hypokinesia, score of 3 for akinesia, score of 4 as

dyskinesia and 5 for aneuysm. So if a patient's echo showed normal (> 45%) left ventricular function, the total Wall motion score was 17 for all segments together. Clinical outcome of the patient was assessed in terms of New York Heart Association (NYHA) classification based on symptoms at first follow up after discharge from the hospital (which was commonly three to four weeks after infarction)

NYHA Grading	Functional Capacity
Class I	No limitations. Ordinary physical activity does not cause undue fatigue, dyspnoea or palpitations (asymptomatic LV dysfunction)
Class II	Slight limitation of physical activity. Ordinary physical activity results in fatigue, palpitation, dyspnoea or angina pectoris (mild CHF).
Class III	Marked limitation of physical activity. Less than ordinary physical activity leads to symptoms (moderate CHF).
Class IV	Unable to carry on any physical activity without discomfort. Symptoms of CHF present at rest (severe CHF).

Statistical Tools

The data collected from the patients was tabulated using Microsoft excel. Statistical analysis was done using SPSS software. Probability was calculated using Chi square test, independent t test and ANOVA test.

P value was considered significant if it was found to be less than 0.05.

REVIEW OF LITERATURE

History

The term electrocardiography is derived from the words electro, kardio and graph which in Greek mean “electrical activity”, “heart” and “to write” respectively[1]. The first clinically relevant invention came in 1901 when Willem Einthoven recorded the ECG using a string galvanometer[2]. He was also responsible for assigning the letters P,Q,R,S & T to the deflections seen in ECG[3].

Later, Einthoven’s work was carried forward by Lewis, who wrote books and articles describing electro-physiology of the heart after conducting various experiments to study dysrhythmias[4]. His work emphasized the benefits of the ECG in the medical fraternity. Williams and James, who were American physicians concluded that the electrocardiogram provided “an entirely new point of view of the normal and morbid action of the heart” after a series of studies[4].

The electrocardiograph, which was called as the string galvanometer for many years underwent a series of transformations over time to become a portable device accessible everywhere[5]. Modern technology allows the application of the same basic principle to the internal cardiac defibrillator and the Holter monitoring system[4].

ST ELEVATION MYOCARDIAL INFARCTION (STEMI)

Definition

According to the American heart association, evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischemia is when the term *acute myocardial infarction (MI)* should be used[6].

The diagnosis of MI is made if any one of the following criteria is fulfilled under these conditions: (Table 1)

TABLE 1

Definition of myocardial infarction

Criteria for acute myocardial infarction

The term acute myocardial infarction (MI) should be used when there is evidence of myocardial necrosis in a clinical setting consistent with acute myocardial ischaemia. Under these conditions any one of the following criteria meets the diagnosis for MI:

- Detection of a rise and/or fall of cardiac biomarker values [preferably cardiac troponin (cTn)] with at least one value above the 99th percentile upper reference limit (URL) and with at least one of the following:
  - Symptoms of ischaemia.
  - New or presumed new significant ST-segment–T wave (ST–T) changes or new left bundle branch block (LBBB).
  - Development of pathological Q waves in the ECG.
  - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
  - Identification of an intracoronary thrombus by angiography or autopsy.
- Cardiac death with symptoms suggestive of myocardial ischaemia and presumed new ischaemic ECG changes or new LBBB, but death occurred before cardiac biomarkers were obtained, or before cardiac biomarker values would be increased.
- Percutaneous coronary intervention (PCI) related MI is arbitrarily defined by elevation of cTn values (>5 x 99th percentile URL) in patients with normal baseline values (≤99th percentile URL) or a rise of cTn values >20% if the baseline values are elevated and are stable or falling. In addition, either (i) symptoms suggestive of myocardial ischaemia or (ii) new ischaemic ECG changes or (iii) angiographic findings consistent with a procedural complication or (iv) imaging demonstration of new loss of viable myocardium or new regional wall motion abnormality are required.
- Stent thrombosis associated with MI when detected by coronary angiography or autopsy in the setting of myocardial ischaemia and with a rise and/or fall of cardiac biomarker values with at least one value above the 99th percentile URL.
- Coronary artery bypass grafting (CABG) related MI is arbitrarily defined by elevation of cardiac biomarker values (>10 x 99th percentile URL) in patients with normal baseline cTn values (≤99th percentile URL). In addition, either (i) new pathological Q waves or new LBBB, or (ii) angiographic documented new graft or new native coronary artery occlusion, or (iii) imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.

Criteria for prior myocardial infarction

Any one of the following criteria meets the diagnosis for prior MI:

- Pathological Q waves with or without symptoms in the absence of non-ischaemic causes.
- Imaging evidence of a region of loss of viable myocardium that is thinned and fails to contract, in the absence of a non-ischaemic cause.
- Pathological findings of a prior MI.

CLINICAL FEATURES

There are three phases used to describe the progression of myocardial infarction. The first phase is acute phase which lasts from the first few hours to seven days. The second phase is the healing phase which last from seven to twenty eight days. This is followed by the healed phase which constitutes the period beyond twenty nine days.

In up to fifty percent of the cases, events like stress (physical or emotional), medical or surgical illness seems to precipitate ST elevation myocardial infarction. The most frequently observed presenting compliant is pain. The characteristic pain of MI is described by patients as heavy, crushing and squeezing.

The site of pain is usually beneath the xiphoid process and epigastrium with radiation to various regions up to occipital area and level of umbilicus. Other associated symptoms include nausea, anxiety, fear of death, sweating, weakness, vomiting etc. Silent infarction appears to be common in the elderly and those with diabetes mellitus.

On examination, patients may appear anxious. Pallor, diaphoresis and cool hands and feet may be observed. Although blood pressure and heart rate may be normal during the first hour of infarction, about fifty percent of those with inferior infarcts manifest parasympathetic

hyperactivity and a quarter of the patients with anterior infarcts can have sympathetic hyperactivity.

On palpation, apical impulse may be difficult to find. In those with anterior infarcts, in the peri-apical area, abnormal systolic pulsations may occur in the initial few days of illness. Signs of ventricular dysfunction like third and fourth heart sounds, paradoxical splitting of S2, reduced intensity of S1 may be present. Other adventitious sounds like pericardial rub, mid or late systolic murmur may be present. The carotid pulse which is representative of the stroke volume can be decreased.

COMPLICATIONS

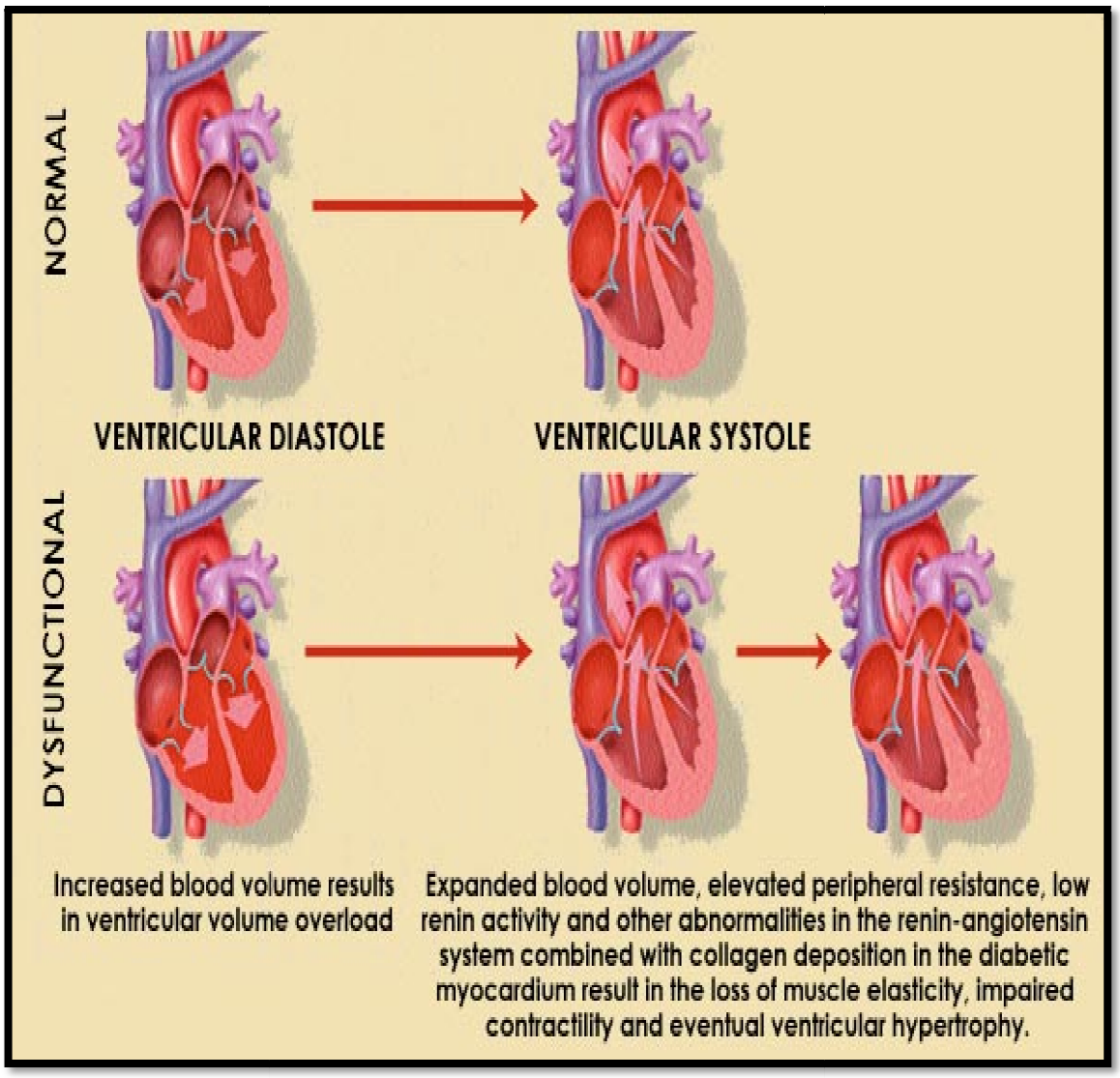
Ventricular Dysfunction

Remodeling of the left ventricle that occurs following STEMI heralds heart failure that may eventually develop months to years after infarction. The left ventricular dilatation that develops after STEMI is because of a combination of acute changes causing elongation of the infarcted area and later because of lengthening of the non-infarcted area(figure 1).

Factors like site and extent of the infarct influence the overall dilatation of the chamber as evidenced by greater dilatation in case of MI involving anterior wall and apex with a graver prognosis. LV dysfunction

serves as the single most important predictor of mortality in STEMI. The dysfunction may be diastolic or systolic. Diastolic dysfunction may manifest as pulmonary venous hypertension and congestion.

FIGURE 1



SYSTOLIC HEART FAILURE: *ventricular function alteration*

Many patients with heart failure can manifest with symptoms and signs suggestive of diminished systolic function of the ventricle. The basic reason behind this cardiac depression is failure or inadequate pump mechanism of the heart which causes insufficiency in optimum cardiac output and maintainence of mean arterial pressure. As the heart failure progresses, systolic function diminishes initially on exertion and later at rest causing limitation in routine activity and affecting the quality of life.

The molecular and cellular basis of systolic heart failure involves not just anomalies in proteins responsible for contraction of myocytes or biochemical abnormalities in calcium homeostasis, but also other mechanisms as well. They include distorted function of channels handling ions, metabolic dysfunction or abnormalities at mitochondrial level, disturbances in structure like myocytolysis and cell loss, electrical remodeling, inflammatory changes like fibrosis or variation in signal transduction[7][8][9].

It is important to know the pathophysiology because it plays a role in therapy targeting these mechanisms to improve systolic function of the heart in a way that is sustainable in terms of long term outcome in addition to combating left ventricular remodeling[10][11].

The evidence of anomalous cardiac cell function can be seen using tissue specimens at molecular level by the study of length and force of myocyte with the membrane removed. But this is challenging in an intact heart in a living being due to various confounding factors like existing loading of the heart, dysfunction due to heart failure, complicated anatomy of the ventricular chamber, remodeling etc. the most widely employed measure of heart failure is determination of the ejection fraction[12]. This is affected by all the above listed confounding factors which makes it a sub-optimal tool for assessment of variable systolic ability of the heart[13].

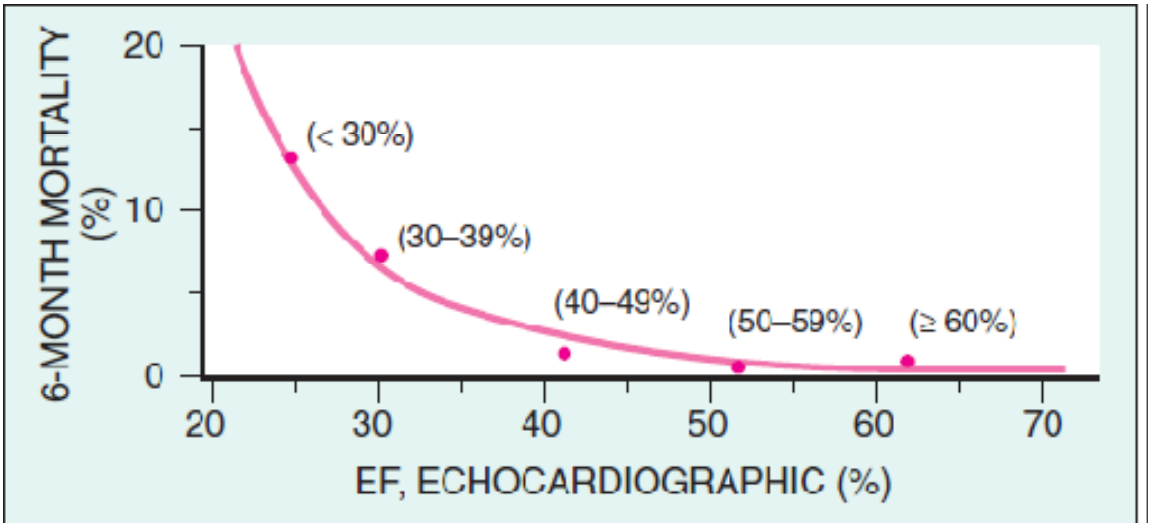
The symptomatic picture may not always correlate with the anatomical remodeling of the heart[12]. For instance, a patient with NYHA class I symptoms preceded by MI can show an ejection fraction of <40% and a patient presenting with class III dyspnea can have dilated cardiomyopathy with a low ejection fraction. In both cases, the fundamental reason behind diminished EF is elevated end diastolic volume secondary to enlargement of the chamber. But the cause for this mechanism is different.

In case of infarction, the main pathology is remodeling due to area of ischemia with possibly a normal reserve capacity representing the rest of the myocardium. In case of the cardiomyopathy, the pathology is

widespread with diminished reserve capacity. In both the instances routine workup and determination of ejection fraction may show similar results. There is a role for invasive pressure volume loop to assess degree of contractile dysfunction[13].

The most important prognosticator of STEMI appears to be left ventricular function[14] (Figure 2). Dysfunction of the left ventricle is of two types - systolic or diastolic. Patients who are affected by myocardial infarction can have either systolic or diastolic dysfunction or both.

FIGURE 2 – Effect of LV function on survival following MI



Hemodynamic Instability

The main cause for death is pump failure and its severity is influenced by the extent of infarction. Based on clinical signs, cardiac

output and wedge pressure, acute MI patients can be categorized into four sub groups(table 2):

TABLE 2

*Based on Clinical Examination**		*Based on Invasive Monitoring†*	
CLASS	**DEFINITION**	**SUBSET**	**DEFINITION**
I	Rales and S_3 absent	I	Normal hemodynamics; PCWP < 18 mm Hg, CI > 2.2
II	Crackles, S_3 gallop, elevated jugular venous pressure	II	Pulmonary congestion; PCWP > 18 mm Hg, CI > 2.2
III	Frank pulmonary edema	III	Peripheral hypoperfusion; PCWP < 18 mm Hg, CI < 2.2
IV	Shock	IV	Pulmonary congestion and peripheral hypoperfusion; PCWP > 18 mm Hg, CI < 2.2

CI = cardiac index; PCWP = pulmonary capillary wedge pressure.
*Modified from Killip T, Kimball J: Treatment of myocardial infarction in a coronary care unit. A two year experience with 250 patients. Am J Cardiol 20:457, 1967.
†Modified from Forrester J, Diamond G, Chatterjee K, et al: Medical therapy of acute myocardial infarction by the application of hemodynamic subsets. N Engl J Med 295:1356, 1976.

Hypovolemia

Various factors can be held responsible for the development of hypotension in STEMI. Hypovolemia can be due to excessive diaphoresis, vomiting, diuretic usage or decreased intake of fluids. In the absence of these factors, with normal essential vascular volume, relative hypovolemia may be present due to decreased ventricular compliance.

Cardiogenic shock

The incidence of Cardiogenic shock has decreased in recent times due to improved treatment measures. Majority of the patients(90%) develop this complication during hospitalization. Cardiogenic shock represents the most severe form of left ventricular damage. Most commonly affected area is the myocardium and in the rest, mechanical defects like ventricular septal or papillary muscle rupture or right ventricular infarction[14].

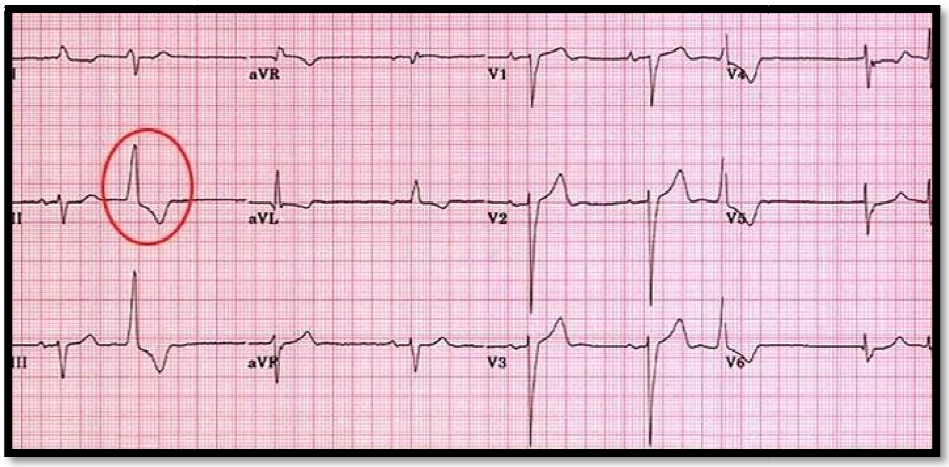
Arrhythmias

The causes of arrhythmias in patients with MI are disturbances of electrolytes, ANS imbalance, delayed conduction in ischemic myocardial zones. Majority of deaths due to arrhythmia occur in first few hours following infarction.

Ventricular Premature Beats

Premature ventricular depolarizations in STEMI (Figure 3) which are sporadic and less frequent do no require therapy. Unless there are ventricular tachyarrhythmias, prophylactic anti-arrhythmic therapy is not needed. Precipitating factors like hypokalemia and hypomagnesemia should be prevented and treated if found to occur.

FIGURE 3 – ECG SHOWING VENTRICULAR PREMATURE COMPLEXES



Ventricular Tachycardia and Ventricular Fibrillation

Paroxysmal and clinically insignificant episodes ventricular tachycardia do not appear to carry risk of death. But the occurrence of sustained ventricular tachycardia (figures 4 & 5) later in the course of the illness, which is common in patients with trans-mural infarction and left ventricular dysfunction, is likely to worsen the outcome.

FIGURE 4 – ECG SHOWING MONOMORPHIC VENTRICULAR TACHYCARDIA

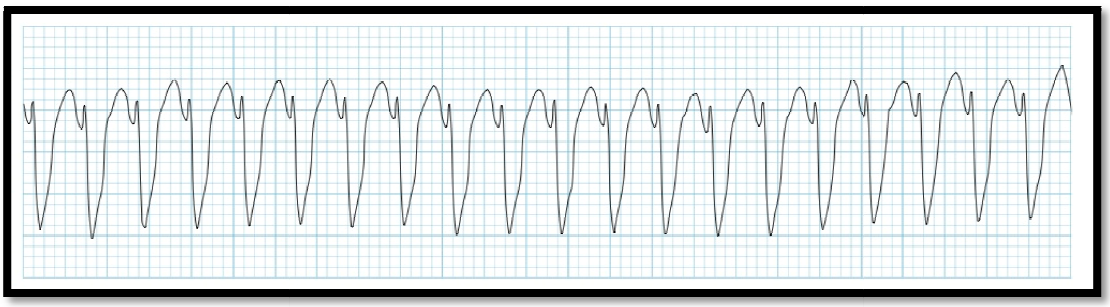
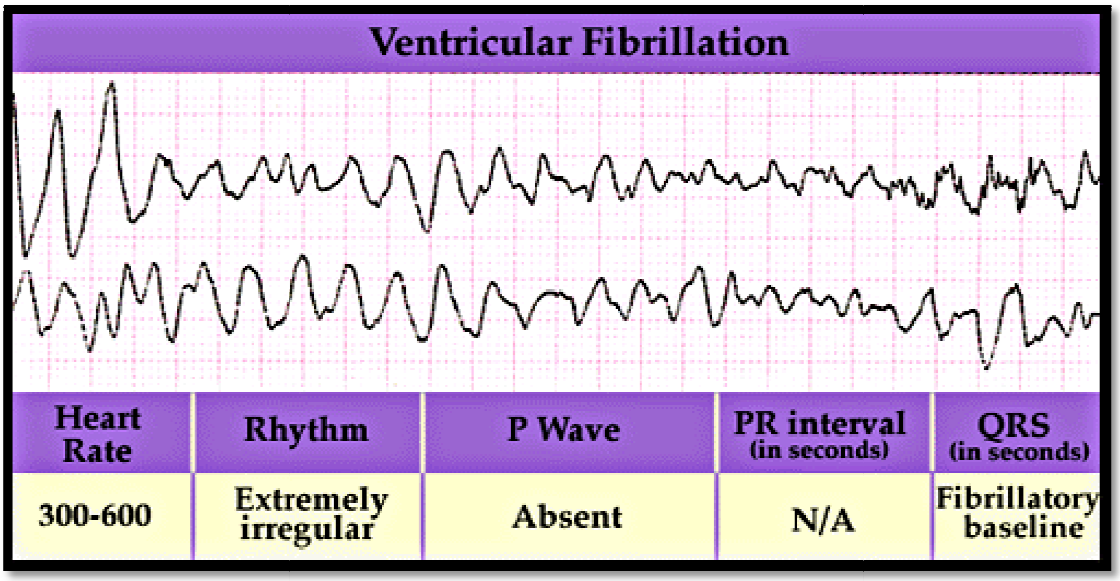


FIGURE 5– ECG SHOWING POLYMORPHIC VENTRICULAR TACHYCARDIA

In patients admitted for STEMI, ventricular fibrillation(VF) (figure 6) may occur in three clinical scenarios. Primary, secondary and late ventricular fibrillation. When the arrhythmia occurs all of a sudden without warning signs of preceding ventricular failure, it is known as primary VF. Data shows that the incidence of primary VF is declining. As the name suggests, secondary VF occurs as a terminal event secondary to worsening left ventricular failure. If it occurs more than forty eight hours or later after STEMI, it constitutes the late type of VF, which is frequently seen in patients with ventricular dysfunction and large infarcts.

FIGURE 6 – ECG SHOWING VENTRICULAR FIBRILLATION

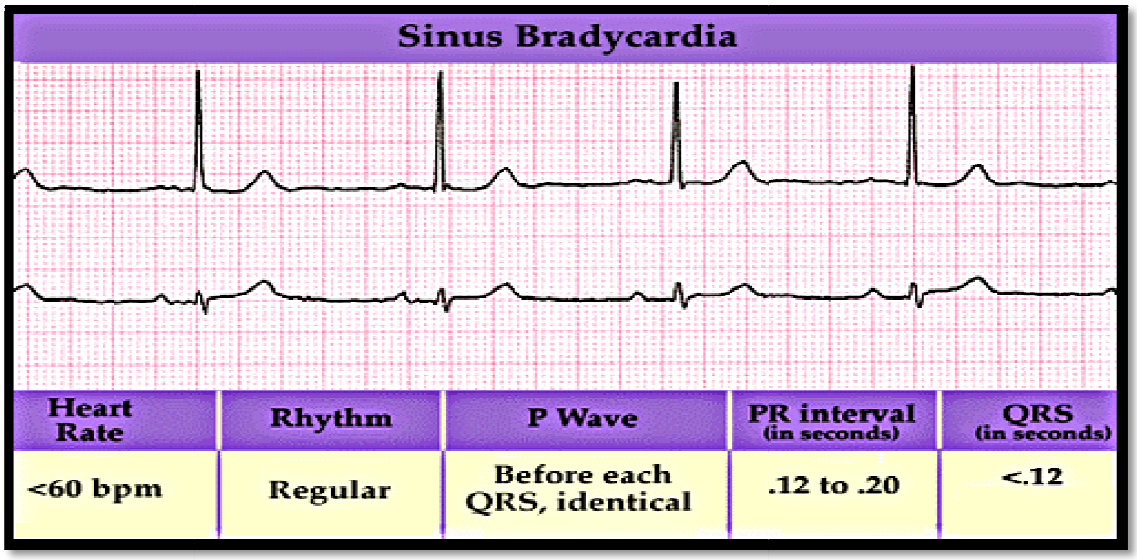


Bradyarrhythmias

Sinus bradycardia

Sinus bradycardia (figure 7) is common during initial phases of STEMI and with inferior and posterior infarction. Treatment is warranted if heart rate falls below forty beats per minute or if there is hemodynamic instability because it is a protective mechanism initiated by vagal tone.

FIGURE 7– ECG SHOWING SINUS BRADYCARDIA



Atrio-ventricular blocks

The ischemic damage in MI can cause conduction defects in atrio-ventricular node or bundle of His. The following table depicts atrio-ventricular conduction disturbances in myocardial infarction(table 3).

TABLE 3 – ATRIOVENTRICULAR CONDUCTION DISTURBANCES IN ACUTE MYOCARDIAL INFARCTION

	Location of Disturbance	
PARAMETER	**PROXIMAL**	**DISTAL**
Site of block	Intranodal	Infranodal
Site of infarction	Inferoposterior	Anteroseptal
Compromised arterial supply	RCA (90%), LCX (10%)	Septal perforators of LAD
Pathogenesis	Ischemia, necrosis, hydropic cell swelling, excess parasympathetic activity	Ischemia, necrosis, hydropic cell swelling
Predominant type of AV nodal block	(a) First-degree (PR > 200 msec) Mobitz type I, second-degree	(a) Mobitz type II, second-degree Third-degree
Common premonitory features of third-degree AV block	(a) First- or second-degree AV block (b) Mobitz I pattern	(a) Intraventricular conduction block (b) Mobitz II pattern
Features of escape rhythm following third-degree block		
(a) Location	(a) Proximal conduction system (His bundle)	(a) Distal conduction system (bundle branches)
(b) QRS width	(b) <0.12/sec*	(b) >0.12/sec
(c) Rate	(c) 45-60/min but may be as low as 30/min	(c) Often <30/min
(d) Stability of escape rhythm	(d) Rate usually stable; asystole uncommon	(d) Rate often unstable with moderate to high risk of ventricular asystole
Duration of high-grade AV block	Usually transient (2-3 days)	Usually transient, but some form of AV conduction disturbance and/or intraventricular defect may persist
Associated mortality rate	Low unless associated with hypotension and/or congestive heart failure	High because of extensive infarction associated with power failure or ventricular arrhythmias
Pacemaker therapy		
(a) Temporary	(a) Rarely required; may be considered for bradycardia associated with left ventricular power failure, syncope, or angina	(a) Should be considered in patients with anteroseptal infarction and acute bifascicular block
(b) Permanent	(b) Almost never indicated because conduction defect is usually transient	(b) Indicated for patients with high-grade AV block with block in His-Purkinje system and those with transient advanced AV block and associated bundle branch block

Intraventricular blocks

Recent studies show the incidence of intra-ventricular blocks as 2% to 5%.

Isolated fasicular block affecting the posterior division carries a higher risk of death as it occurs as a consequence of larger infarcts.

Right bundle branch block in anterior STEMI in the setting of congestive heart failure is associated with markedly elevated mortality

risk. Usually, it is a new onset lesion and may frequently cause a predisposition to ventricular fibrillation in hospitalized patients.

Bifasicular block poses a risk for development of complete atrio ventricular block. Extensive myocardial infarction is needed to cause such a conduction defect. Therefore, mortality rate is higher in such cases due to extensive pump failure.

Supraventricular Tachyarrhythmias

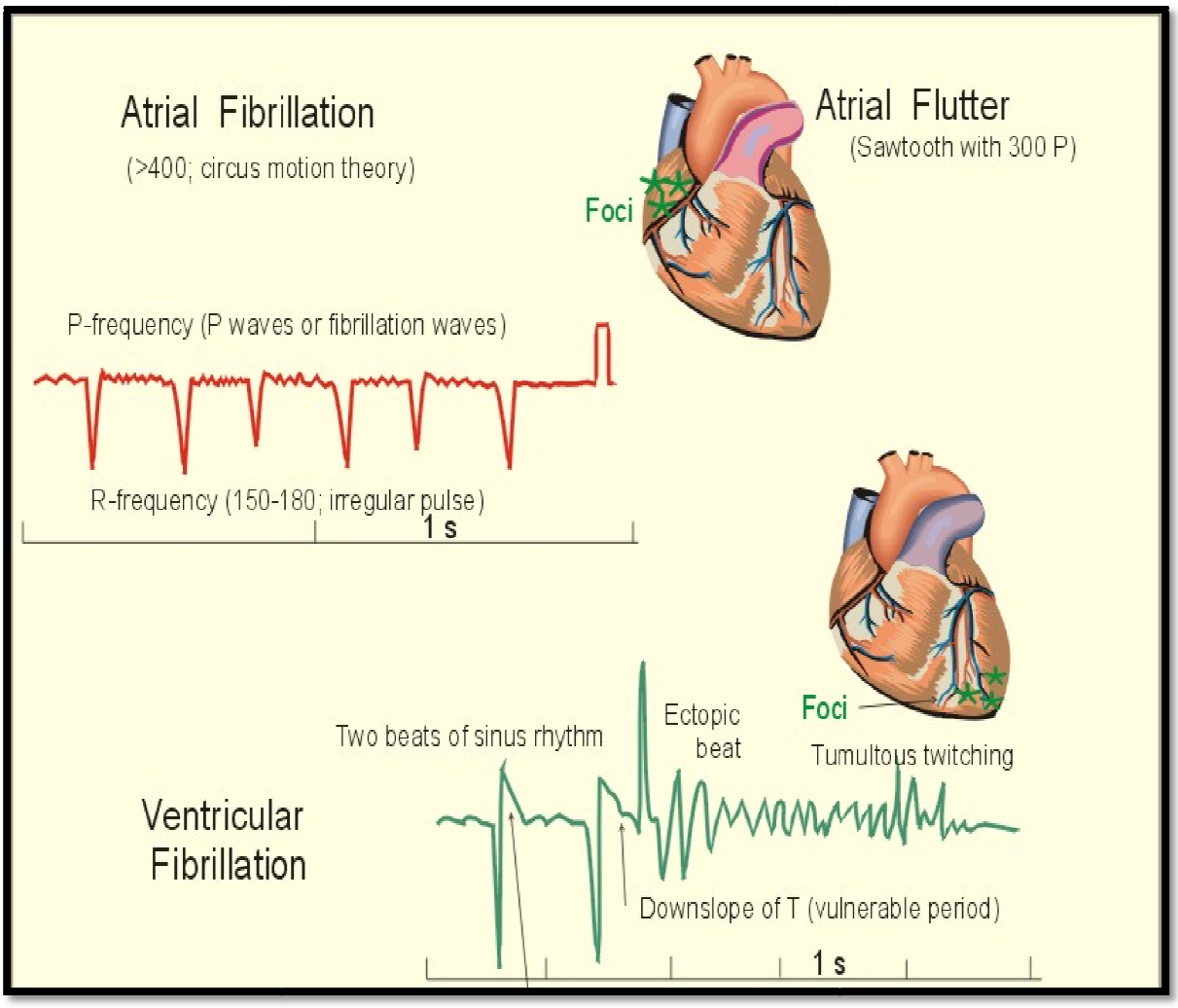
Sinus tachycardia

The clinical features associated with MI like pain, anxiety, pericarditis, left ventricular failure, hypovolemia and pharmacotherapy with atropine or inotropes cause sympathetic overactivity which lead to sinus tachycardia. It commonly occurs in conjunction with anterior wall infarcts and left ventricular dysfunction. Continuing heart failure is represented by persisting sinus tachycardia which carries a poorer outcome. Beta blockers are indicated in sinus tachycardia if the causative factor of the arrhythmia is hyperdynamic circulation. This is applicable to young patients with first time STEMI. Contraindication to beta blocker usage is hemodynamic instability in the form of left ventricular dysfunction or hypotension.

Atrial Flutter and Fibrillation

Atrial sympathetic stimulation can cause ephemeral atrial flutter (figure 8) in STEMI especially in those with pulmonary emboli or left ventricular failure and invariably poses a risk of deterioration due to its adverse effect on hemodynamic stability.

FIGURE 8 – showing atrial fibrillation & flutter



In addition to the predisposing causes for flutter, Atrial fibrillation (figure 8) can be precipitated by pericarditis, right ventricular infarction and atrial ischemic injury. In STEMI, atrial fibrillation is associated with stroke and increased mortality especially in anterior wall MI. It is a sign of poor prognosis.

Other complications

- *Recurrent chest discomfort* may occur in about 25% of patients admitted for STEMI with a higher prevalence in post-thrombolysis cases. Patients who experience recurrent angina should ideally undergo coronary angiography because it may signify new infarct or extension of the previous infarct and worsens the risk of death by three fold.

- *Dressler syndrome* can occur during one to eight weeks post infarction, thus giving it its name postmyocardial infarction syndrome. Clinical features include fever, malaise, pericardial pain, elevated white blood cell count & erythrocyte sedimentation rate and pericardial effusion. Treatment is with large doses of aspirin (650 mg can be given with a frequency of four hours).

- *Pericarditis* can occur in patients who have epicardial involvement in STEMI as manifested by friction rubs or pain. It is important to distinguish the pericardial pain from ischemic pain to avoid unnecessary use of anti-coagulants which can lead to tamponade.

- *Thromboembolism* - 10% of complications in STEMI are due to clinically significant thromboembolisms. This percentage of silent lesions is higher. Precipitant factors include extensive anterior infarction,left ventricular thrombus (as in echo) and congestive cardiac failure. Related complications comprise of hemiparesis and hypertension when cerebral and renal vasculature is involved respectively.

- *Left ventricular aneurysm* may cause sequelae like heart failure, emboli and arrhythmias. Clinical features of an aneurysm include displaced or double localized apical impulse. Echo serves as a valuable tool for diagnosis. Rupture is rare.

The following table shows the management of arrhythmias in MI (table 4):

TABLE 4 – SHOWING MANAGEMENT OF ARRHYTHMIAS DURING MI

CATEGORY	ARRHYTHMIA	OBJECTIVE OF TREATMENT	THERAPEUTIC OPTIONS
Electrical instability	Ventricular premature beats	Correction of electrolyte deficits and increased sympathetic tone	Potassium and magnesium solutions, beta blocker
	Ventricular tachycardia	Prophylaxis against ventricular fibrillation, restoration of hemodynamic stability	Antiarrhythmic agents; cardioversion/ defibrillation
	Ventricular fibrillation	Urgent reversion to sinus rhythm	Defibrillation; bretylium tosylate
	Accelerated idioventricular rhythm	Observation unless hemodynamic function is compromised	Increase sinus rate (atropine, atrial pacing); antiarrhythmic agents
	Nonparoxysmal atrioventricular junctional tachycardia	Search for precipitating causes (e.g., digitalis intoxication); suppress arrhythmia only if hemodynamic function is compromised	Atrial overdrive pacing; antiarrhythmic agents; cardioversion relatively contraindicated if digitalis intoxication present
Pump failure, excessive sympathetic stimulation	Sinus tachycardia	Reduce heart rate to diminish myocardial oxygen demands	Antipyretics; analgesics; consider beta blocker unless congestive heart failure present; treat latter if present with anticongestive measures (diuretics, afterload reduction)
	Atrial fibrillation and/or atrial flutter	Reduce ventricular rate; restore sinus rhythm	Verapamil, digitalis glycosides; anticongestive measures (diuretics, afterload reduction); cardioversion; rapid atrial pacing (for atrial flutter)
	Paroxysmal supraventricular tachycardia	Reduce ventricular rate; restore sinus rhythm	Vagal maneuvers; verapamil, cardiac glycosides, beta-adrenergic blockers; cardioversion; rapid atrial pacing
Bradyarrhythmias and conduction disturbances	Sinus bradycardia	Acceleration of heart rate only if hemodynamic function is compromised	Atropine; atrial pacing
	Junctional escape rhythm	Acceleration of sinus rate only if loss of atrial "kick" causes hemodynamic compromise	Atropine; atrial pacing
	Atrioventricular block and intraventricular block		Insertion of pacemaker

Modified from Antman EM, Rutherford JD (eds): Coronary Care Medicine: A Practical Approach. Boston, Martinus Nijhoff, 1986, p 78.

ELECTROCARDIOGRAM

The heart's principle function is to supply oxygen rich blood into systemic circulation. By rhythmically contracting it pumps blood from lungs to systemic circulation.

The contraction of heart is enabled by signaling via electrical impulses through cardiac muscle. The currents are initiated by pace maker cells and propogated via specific conduction tissues and cardiac muscle itself[15].

Cardiac Electrical Activation

Electrical impulses start in sinoatrial(SA) node (pacemaker) where it is self generated automatically and propagated to right and then left atrium. The electrical signals then passes through improvised conduction tissue in atrio-ventricular(AV) junction. Between the atria and the ventricles the AV junction acts as a relay junction with the proximal portion termed as AV node and distal portion termed bundle of His.

The electrical impulse reaches the left and right ventricular myocardium from bundle of His through left and right bundle branches and finally Purkinje fibres. Electromechanical coupling is the electrically stimulated commencement of cardiac contraction aided by release of ions of calcium by the cardiac muscle cells of atrium and ventricles.

The electrical activity propagating through the heart(from instant to instant) recorded on a specialized graph is called as the electrocardiogram (ECG) recorded with the aid of an electrocardiograph machine. Currents produced by only the working heart muscle is recorded by the ECG. The smaller currents produced by AV, SA nodes are invisible. The ECG is recorded by placement of electrodes from chest, arms and legs of a person. The standard ECG consists of twelve leads which show variations in voltage among electrodes on surface of body.

The twelve leads consist of 6 precordial (chest)(figure 10) and 6 extremity (limb) leads. The purpose of the limb leads and the extremity leads is to record voltages transmitted onto the frontal plane and horizontal plane of the body respectively[16].

The extremity leads are recorded by placement of electrodes on both the wrists and both ankles where the right leg electrode acts as electrical ground. The heart voltages are relayed through trunk to limbs. Of these extremity leads, three are bipolar leads (I, II, III) (figure 9) and unipolar augmented leads (aVR, aVL & aVF). The difference in voltages between left arm and right arm is recorded by lead I.

The difference in voltages between left leg and right arm is recorded by lead II. The difference in voltages between left leg and left arm is recorded by lead III. Augmented leads record voltages at one point in realation to another electrode nearer to zero potential. The axis and orientation of leads in relation to the heart is shown in figure 11.

FIGURE 9 – SHOWING EINTHOVEN'S TRIANGLE

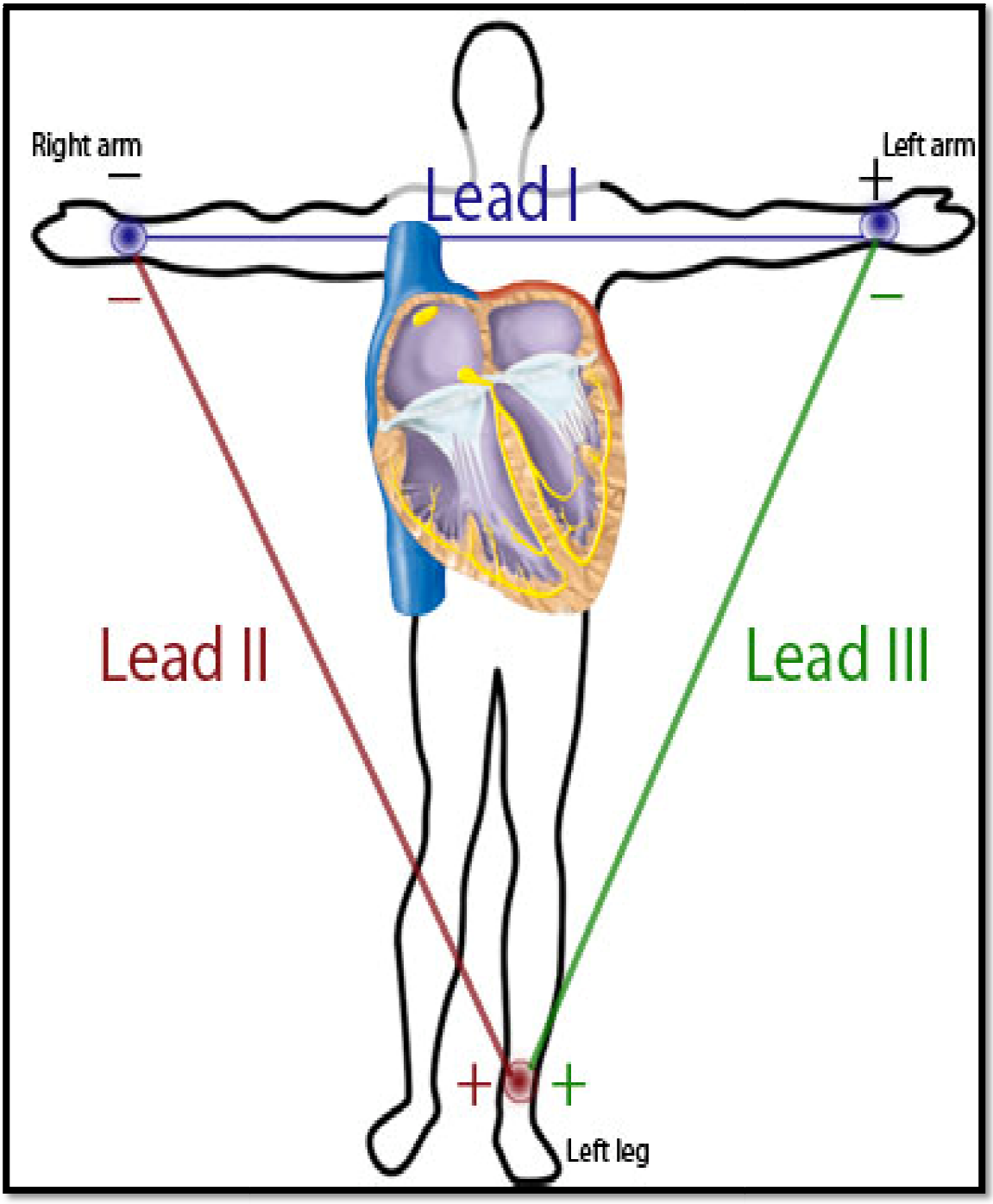


FIGURE 10– SHOWING PRECORDIAL LEAD PLACEMENT

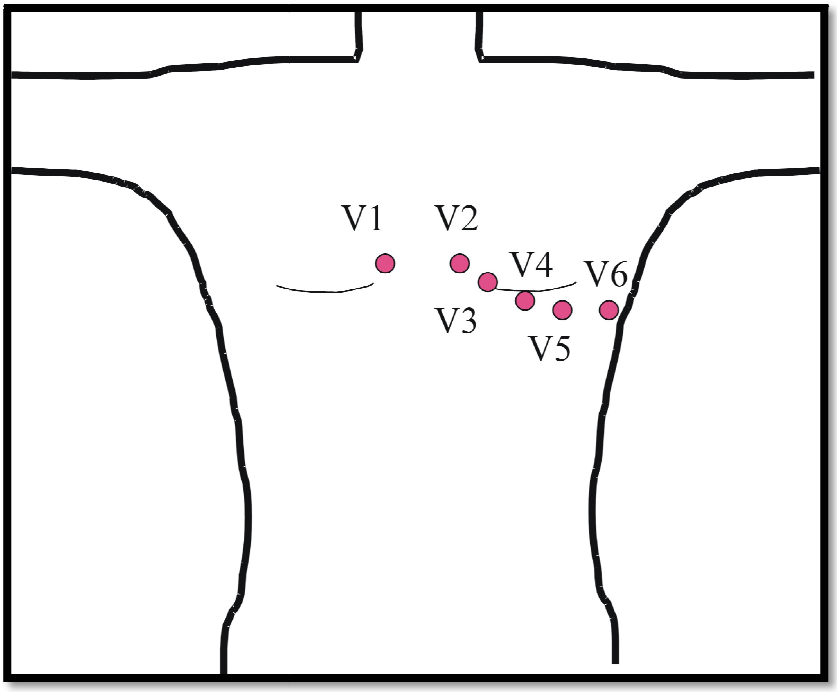
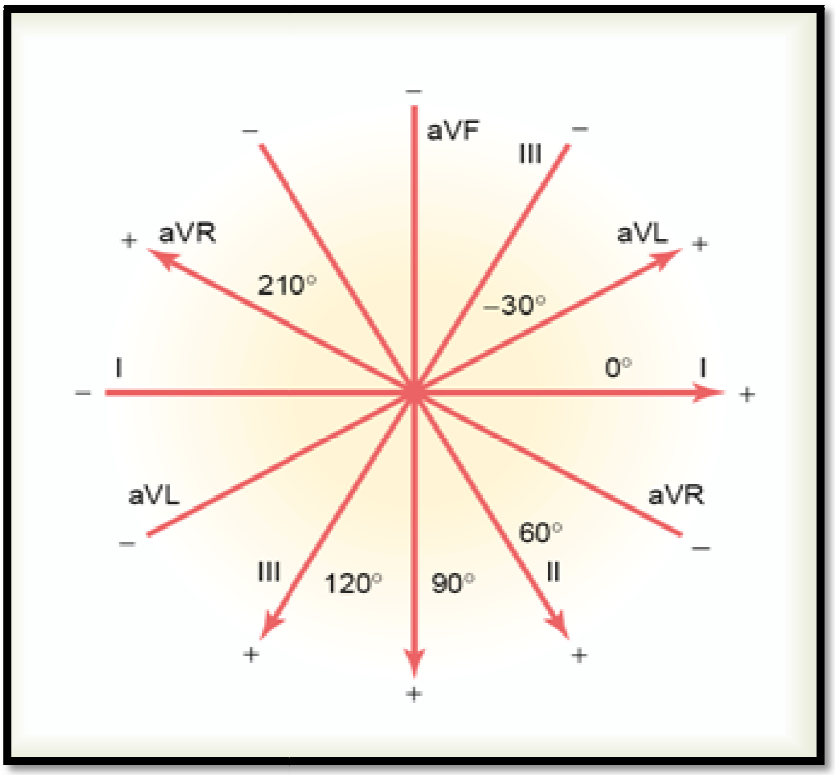
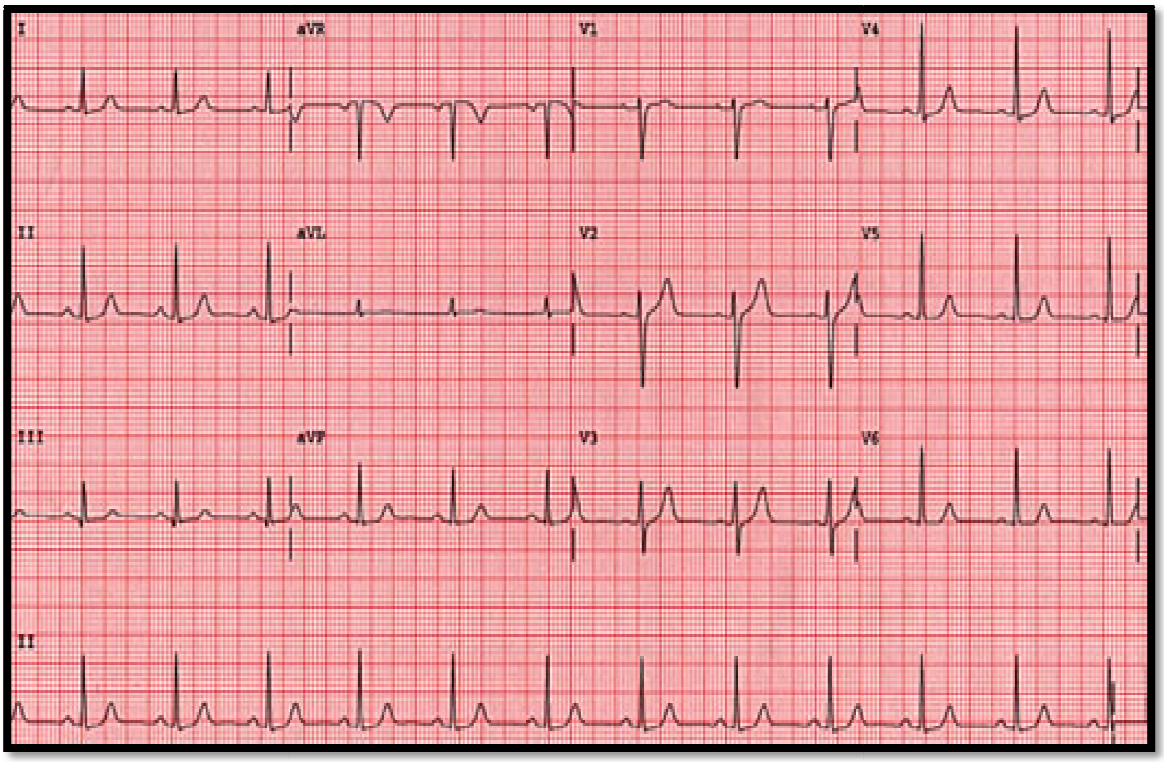


FIGURE 11 - ECG LEAD ORIENTATION & AXIS



The stimulus which is spread through the cardiac muscle resulting in cardiac muscle cell stimulation is called as depolarization. The activated cardiac muscle reverts back to resting state which is called as repolarization.

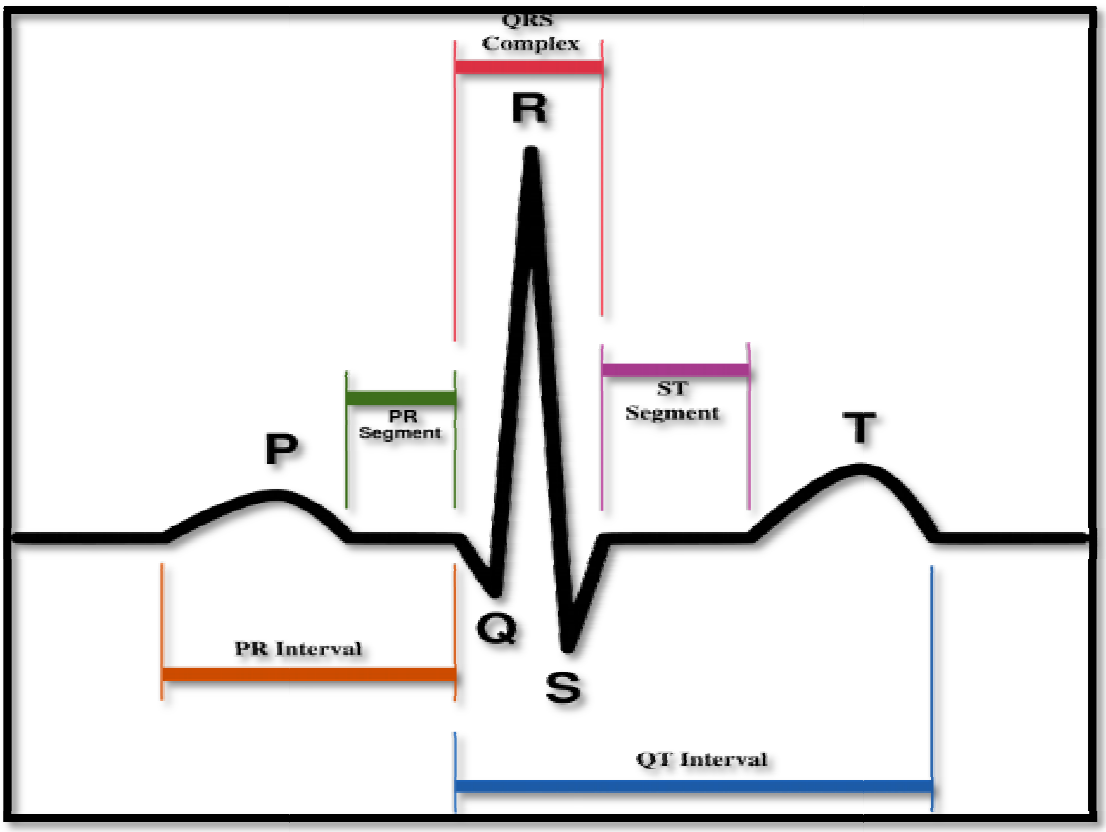
FIGURE 12- STANDARD ECG GRAPH PAPER showing the leads



The various deflections are recorded on a special paper which consists of grid like boxes and its called an ECG graph paper (figure 12). A small box has an area of 1 mm^2 and the standard ECG graph paper speed is 25 mm/second, hence each small unit constitutes 0.04 second horizontally. In the vertical direction, the standard ECG machine is

calibrated to show a deflection of 10 mm amplitude for every 1 mV of electrical impulse transmitted[15]. A wave is called positive if it has an upward deflection and negative in case of a downward deflection.

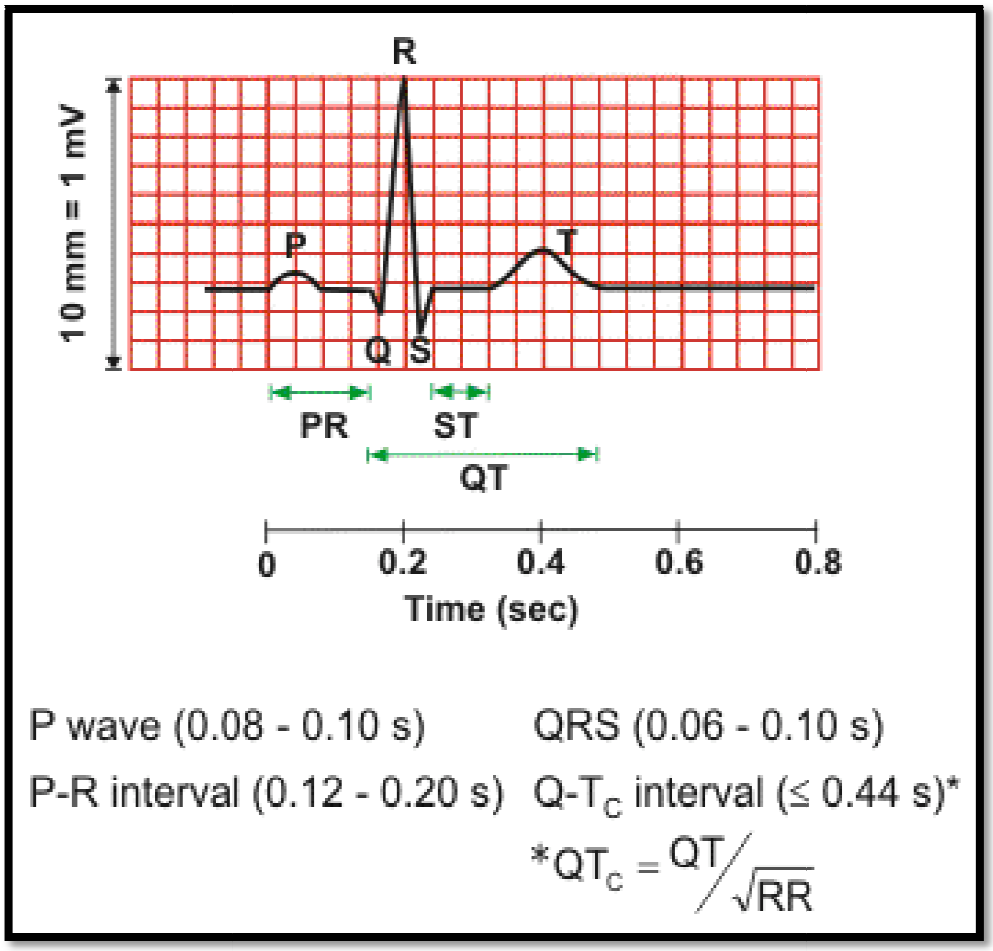
FIGURE 13 - SHOWING COMPONENTS OF ECG



The standard ECG consists of P wave, QRS complex, ST segment, T wave and U wave (figure 13). The P wave represents the atrial depolarization. The QRS complex is representative of ventricular depolarization. The ST segment, T wave & U wave represent the ventricular repolarization with U wave being the terminal phase. The time required for the propagation of electric impulses via the AV junction is

represented in the ECG as the PR interval which is the distance between the start of the P wave and the start of QRS complex. The normal PR interval in an adult is 0.1 – 0.2 seconds(Figure 14).

FIGURE 14- SHOWING DURATION OF ECG COMPONENTS



QRS Complex

The QRS complex denotes the dispersion of electrical impulses in the ventricles. The first negative deflection in the QRS complex is called the Q wave and positive deflection is called R wave. The subsequent negative deflection is called S wave. In certain conditions the QRS

complex is either completely positive or completely negative where it is called as R wave and QS wave respectively.

The time interval taken for the electrical impulse to pass through the ventricles is known by measuring the QRS width. The normal time interval is 0.1 second.

The characteristics of the QRS complex varies in different leads according to the way they are placed in relation to the heart, which in common language can be called the way the respective leads “look” at the heart[15]. The QRS complex, normally is positive (which means above the baseline) in apico-lateral leads because they are oriented in such a way that they look at the heart from the left side. The same applies to the leads that look at the heart from below, as from under the heart (II,III & aVF). But if the QRS complex is negative in the leads V1,V2,aVR which record the electrical activity of the heart from the right side.

The QRS complex will be partly above and partly below the baseline (known as biphasic), in the anterior leads like V3 & V4. The amplitude of the QRS complex is usually not more than 25 mm.

QRS AMPLITUDE

Prominent variation can occur in the amplitude and morphology of the QRS complex due to certain influencing factors like age, gender and ethnicity[17][18][19,20]. The amplitude is inversely proportional to age, but it is said that the variation is less significant after forty years of age. Females tend to have a lower amplitude than males and Caucasians have lower amplitudes than those of African American ethnicity[21].

The following tables depict the results of the study showing the variability of the amplitude in different waves of the QRS complex in terms of gender differences and across different leads of ECG[22](tables 5 &6).

TABLE 5

Lead	Age (Years)	Q Wave	R Wave	S Wave	T Wave
I	12–16	1.0 (0–3.0)	5.9 (0–12.0)	1.3 (0–10.0)	2.4 (1.6–6.0)
	16–20	0.3 (0–1.3)	4.3 (1.8–9.5)	1.0 (0–3.5)	2.1 (0.2–3.7)
	20–30 ♂	0.3 (0–2.6)	5.7 (1.3–12.9)	1.3 (0–4.0)	2.1 (0.9–4.1)
	20–30 ♀	0.1 (0–1.0)	4.8 (1.3–9.5)	0.8 (0–3.2)	2.1 (0.5–4.0)
	30–40 ♂	0.2 (0–1.2)	5.4 (1.8–11.3)	1.2 (0–4.2)	2.0 (0.6–3.7)
	30–40 ♀	0.2 (0–1.1)	5.1 (1.4–15.0)	0.6 (0–2.9)	2.0 (0.9–3.8)
	40–60 ♂	0.2 (0–1.1)	6.0 (2.0–11.6)	0.7 (0–3.0)	1.9 (0.7–3.5)
	40–60 ♀	0.2 (0–1.2)	6.2 (2.0–12.5)	0.3 (0–2.1)	1.9 (0.7–3.2)
II	12–16	1.0 (0–2.5)	13.5 (3.5–24.5)	1.4 (0–7.0)	3.3 (–0.2 to +6.1)
	16–20	0.5 (0–2.8)	9.5 (2.9–15.8)	1.4 (0–6.3)	2.7 (0.2–5.7)
	20–30 ♂	0.5 (0–2.1)	11.7 (4.7–19.1)	1.4 (0–4.8)	2.9 (0.8–5.8)
	20–30 ♀	0.3 (0–2.2)	9.9 (3.9–15.9)	0.6 (0–2.9)	2.4 (0.7–4.4)
	30–40 ♂	0.3 (0–1.3)	9.3 (4.0–17.0)	1.3 (0–4.3)	2.7 (1.0–5.0)
	30–40 ♀	0.2 (0–1.3)	8.7 (2.1–15.5)	0.8 (0–3.2)	2.2 (0.6–4.9)
	40–60 ♂	0.3 (0–1.2)	7.5 (1.9–15.5)	0.8 (0–3.8)	2.3 (0.8–4.3)
	40–60 ♀	0.2 (0–1.5)	8.1 (2.6–15.3)	0.7 (0–3.1)	2.2 (0.8–4.1)
III	12–16	1.6 (0–5.0)	9.0 (1.0–26.0)	1.1 (0–9.0)	0.8 (–1.6 to +3.5)
	16–20	0.6 (0–4.6)	6.8 (1.2–15.0)	1.1 (0–4.9)	0.8 (–1.9 to +3.9)
	20–30 ♂	0.6 (0–2.6)	7.1 (0.8–15.8)	1.1 (0–6.0)	0.8 (–0.9 to +3.0)
	20–30 ♀	0.6 (0–2.2)	6.0 (0.5–14.2)	0.6 (0–4.3)	0.3 (–0.7 to +2.1)
	30–40 ♂	0.5 (0–2.0)	5.0 (0.3–12.4)	1.4 (0–8.5)	0.7 (–1.5 to +2.9)
	30–40 ♀	0.3 (0–1.0)	4.5(0.1–12.7)	0.8 (0–4.5)	0.4 (–1.8 to +2.0)
	40–60 ♂	0.4 (0–2.4)	3.2 (0.1–11.9)	1.6 (0–7.5)	0.4 (–1.2 to +2.2)
	40–60 ♀	0.4 (0–2.0)	3.6 (0.1–11.8)	1.4 (0–6.0)	0.3 (–1.1 to +1.7)
aV_R	12–16	7.9 (0–14.0)	1.2 (0–3.0)	2.5 (0–19.0)	–2.9 (–5.2 to –0.5)
	16–20	2.1 (0–9.0)	1.2 (0–4.7)	4.3 (0–11.1)	–2.0 (–4.8 to –0.1)
	20–30 ♂	2.5 (0–11.5)	0.6 (0–3.2)	9.0 (0–16.1)	–2.5 (–5.0 to –0.8)
	20–30 ♀	2.5 (0–11.5)	0.5 (0–1.8)	6.9 (0–11.6)	–2.2 (–5.0 to –0.7)
	30–40 ♂	2.1 (0–9.0)	0.6 (0–3.2)	7.6 (0–12.0)	–2.3 (–4.2 to –1.0)
	30–40 ♀	2.1 (0–9.0)	0.5 (0–2.2)	6.2 (0–14.6)	–2.1 (–3.5 to –0.9)
	40–60 ♂	2.0 (0–8.5)	0.5 (0–2.2)	6.8 (0–11.0)	–2.1 (–3.4 to –0.8)
	40–60 ♂	2.0 (0–8.5)	0.4 (0–1.6)	6.8 (0–12.5)	–2.0 (–3.6 to –0.7)
aV_L	12–16	1.0 (0–6.0)	2.4 (0–12.0)	3.0 (0–20.0)	1.1 (–1.0 to +3.6)
	16–20	0.5 (0–2.5)	1.9 (0.2–6.0)	2.0 (0–8.0)	0.6 (–1.8 to +3.6)
	20–30 ♂	0.3 (0–3.5)	2.0 (0.1–8.3)	2.7 (0–8.7)	1.7 (–0.8 to +2.1)
	20–30 ♀	0.3 (0–3.5)	1.9 (0.1–7.2)	2.0 (0–9.2)	1.0 (–0.4 to +2.2)
	30–40 ♂	0.3 (0–2.3)	2.4 (0.1–8.5)	1.8 (0–6.8)	1.8 (–1.0 to +2.1)
	30–40 ♀	0.1 (0–1.3)	2.3 (0.3–9.2)	1.0 (0–4.5)	1.0 (–0.4 to +3.0)
	40–60 ♂	0.2 (0–1.3)	3.4 (0.2–9.3)	1.1 (0–4.6)	1.1 (–0.5 to +2.2)
	40–60 ♀	0.2 (0–1.4)	3.3 (0.2–8.5)	0.7 (0–3.9)	0.7 (–0.1 to +2.6)
aV_F	12–16	1.3 (0–3.0)	10.2 (0.1–21.8)	1.0 (0–4.0)	2.3 (–0.7 to +5.4)
	16–20	0.8 (0–3.8)	7.7 (1.8–14.0)	1.0 (0–4.9)	1.8 (–0.6 to +5.2)
	20–30 ♂	0.5 (0–2.2)	8.8 (1.0–16.9)	0.7 (0–4.9)	1.1 (–0.2 to +3.5)
	20–30 ♀	0.3 (0–1.6)	7.6 (1.8–15.6)	1.0 (0–2.1)	0.5 (–0.1 to +3.1)
	30–40 ♂	0.3 (0–1.3)	6.7 (1.0–14.8)	0.8 (0–4.4)	1.0 (0.4–3.8)
	30–40 ♀	0.3 (0–1.2)	6.2 (1.0–13.9)	1.2 (0–2.6)	0.5 (0.1–3.2)
	40–60 ♂	0.2 (0–1.2)	4.7 (0.3–12.6)	0.9 (0–4.1)	0.9 (0–3.1)
	40–60 ♀	0.2 (0–11)	5.3 (0.3–13.4)	1.0 (0–3.9)	0.8 (–0.2 to +2.6)
V_1	12–16	0	5.6 (0–16.0)	13.8 (5.0–26.0)	–1.5 (–4.0 to +7.0)
	16–20	0	4.6 (0.4–16.7)	11.7 (1.8–25.1)	0.9 (–3.5 to +6.0)
	20–30 ♂	0	3.3 (0.3–8.9)	11.4 (5.0–18.0)	0.9 (–2.2 to +3.9)
	20–30 ♀	0	1.6 (0–5.3)	7.4 (1.6–14.2)	–0.7 (–2.1 to +2.0)
	30–40 ♂	0	2.2 (0.2–5.4)	9.1 (3.2–17.6)	0.7 (–1.4 to +3.3)
	30–40 ♀	0	1.6 (0–5.8)	7.6 (3.8–14.3)	–0.6 (–2.6 to +1.2)
	40–60 ♂	0	1.7 (0.1–4.9)	8.6 (2.9–16.7)	0.9 (–1.3 to +3.9)
	40–60 ♀	0	1.4 (0.1–4.0)	7.2 (2.3–15.1)	–0.2 (–1.9 to +1.5)

TABLE 6

Lead	Age (Years)	Q Wave	R Wave	S Wave	T Wave
V_2	12–16	0	9.1 (2.0–21.0)	20.1 (4.0–51.0)	4.5 (–4.3 to +13.5)
	16–20	0	7.3 (0.5–20.5)	16.2 (2.6–45.5)	3.9 (–3.8 to +14.1)
	20–30 ♂	0	7.4 (1.7–13.9)	18.0 (6.7–29.1)	6.5 (1.1–12.3)
	20–30 ♀	0	4.6 (1.1–9.2)	12.4 (4.0–23.2)	3.1 (0–4.1)
	30–40 ♂	0	5.4 (0.6–12.1)	15.2 (6.1–27.8)	6.2 (2.0–11.1)
	30–40 ♀	0	3.7 (0.4–10.1)	11.3 (4.2–19.8)	2.9 (0–7.5)
	40–60 ♂	0	4.6 (0.6–12.0)	12.7 (5.2–23.3)	5.5 (1.7–10.1)
	40–60 ♀	0	3.6 (0.2–9.1)	9.4 (2.4–18.0)	3.0 (0.1–6.5)
V_3	12–16	0.1 (0–1)	11.8 (2.0–33.0)	14.1 (3.0–34.0)	4.1 (0–13.0)
	16–20	0.1 (0–1)	8.5 (1.6–23.3)	10.7 (0.9–28.9)	5.1 (–3.7 to +13.5)
	20–30 ♂	0 (0–0.4)	11.6 (2.2–26.6)	10.6 (6.7–22.0)	6.5 (1.9–11.7)
	20–30 ♀	0 (0–0.4)	8.2 (2.3–17.5)	6.1 (4.0–14.2)	3.5 (0–8.6)
	30–40 ♂	0 (0–0.5)	9.4 (2.2–22.5)	10.0 (6.1–22.0)	6.3 (3.1–11.5)
	30–40 ♀	0 (0–0.5)	7.1 (0.8–23.3)	5.1 (4.2–11.9)	3.1 (0.5–7.7)
	40–60 ♂	0 (0–0.4)	8.4 (1.4–11.6)	9.8 (5.2–19.0)	6.0 (2.1–10.7)
	40–60 ♀	0 (0–0.4)	7.1 (1.0–17.7)	6.0 (2.4–13.5)	3.4 (0.1–7.4)
V_4	12–16	0.5 (0–3.0)	23.5 (5.0–51.0)	7.0 (1.0–30.0)	7.2 (0–17.2)
	16–20	0.1 (0–1.0)	12.7 (3.1–30.1)	6.3 (0.2–15.0)	4.7 (–3.6 to +12.6)
	20–30 ♂	0.3 (0–2.9)	16.6 (6.1–27.7)	6.1 (0–15.0)	5.6 (1.5–11.8)
	20–30 ♀	0.1 (0–0.7)	11.5 (5.0–19.6)	2.9 (0–8.5)	3.6 (1.0–7.8)
	30–40 ♂	0.2 (0–1.7)	14.8 (5.2–29.2)	5.7 (1.1–12.1)	5.4 (2.0–9.9)
	30–40 ♀	0.2 (0–1.4)	11.8 (4.1–25.9)	2.4 (0–7.8)	3.3 (0.8–7.0)
	40–60 ♂	0.1 (0–1.0)	14.2 (5.2–25.6)	6.3 (0.8–14.1)	5.4 (1.6–10.4)
	40–60 ♀	0.2 (0–1.3)	12.4 (3.7–23.6)	2.8 (0–7.7)	3.5 (1.0–6.3)
V_5	12–16	1.3 (0–4.0)	18.2 (5.0–35.0)	2.5 (0–12.0)	5.7 (0.5–11.5)
	16–20	0.5 (0–2.8)	11.4 (4.1–26.5)	2.2 (0–8.1)	3.8 (0.2–10.6)
	20–30 ♂	0.7 (0–3.1)	15.3 (5.9–24.0)	2.2 (0–6.4)	3.8 (0.8–8.1)
	20–30 ♀	0.3 (0–1.2)	11.5 (5.2–18.7)	1.0 (0–4.0)	3.0 (1.0–5.5)
	30–40 ♂	0.4 (0–2.0)	14.3 (8.1–24.8)	2.3 (0–6.7)	3.7 (1.3–7.0)
	30–40 ♀	0.3 (0–1.8)	11.8 (5.0–27.2)	0.8 (0–3.2)	2.9 (0.8–5.9)
	40–60 ♂	0.3 (0–1.6)	14.1 (5.9–25.0)	2.4 (1.0–6.9)	3.9 (1.3–7.8)
	40–60 ♀	0.3 (0–1.2)	12.4 (5.0–20.9)	1.0 (0–5.0)	2.9 (0.9–5.1)
V_6	12–16	1.3 (0–2.5)	12.5 (4.0–27.0)	1.0 (0–6.0)	4.0 (0.8–7.2)
	16–20	0.6 (0–4.2)	13.5 (7.0–21.0)	1.2 (0–5.0)	3.8 (0.8–7.1)
	20–30 ♂	0.7 (0–2.6)	11.6 (3.7–19.3)	0.9 (0–3.7)	2.6 (0.5–5.9)
	20–30 ♀	0.4 (0–1.8)	9.6 (5.2–16.3)	0.3(0–1.8)	2.4 (0.9–5.0)
	30–40 ♂	0.5 (0–1.6)	10.9 (5.9–18.3)	0.8 (0–2.8)	2.5 (0.8–4.5)
	30–40 ♀	0.3 (0–1.5)	9.2 (4.0–20.2)	0.3 (0–1.7)	3.3 (0.6–4.7)
	40–60 ♂	0.4 (0–1.5)	10.5 (4.9–17.8)	0.7 (0–2.9)	2.6 (0.8–4.9)
	40–60 ♀	0.3 (0–1.4)	9.6 (3.6–16.8)	0.3 (0–2.6)	2.3 (0.7–4.6)

Adapted from Lepeschkin E. *In:* Altman PE, Dittmer DS (eds): Respiration and Circulation. Bethesda, Md, Federation of American Societies for Experimental Biology, 1971, p 277.

♂ = male; ♀ = female.

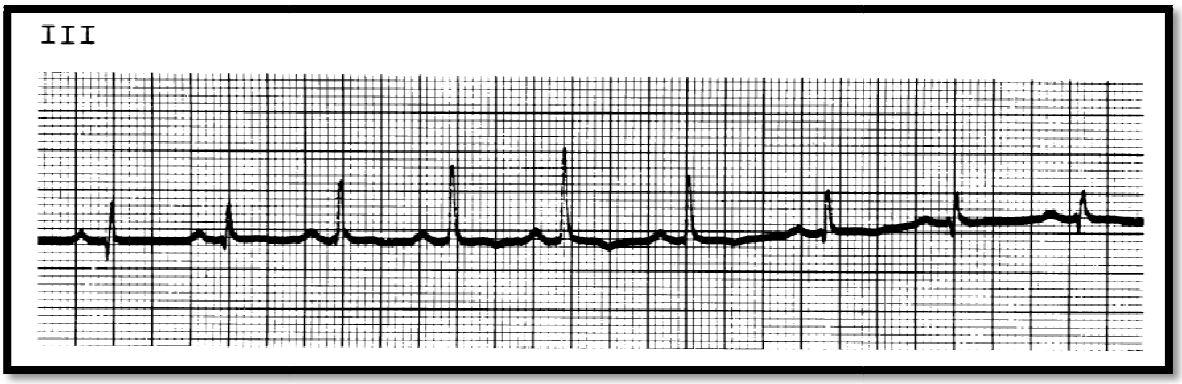
Results are given as means, with the ranges in parentheses.

QRS MORPHOLOGY IN LIMB LEADS

The vector orientation and amplitude affects the morphology of QRS complex in the limb leads. In lead I , a prominent R wave may be seen as the dominant deflection. The whole QRS complex can be negative in aVL. In limb lead II, if there is a normal axis , dominant R wave will be present. But in the lead aVR which faces towards the cavity of the heart, negative wave predominates[21].

A major positive deflection is commonly seen in aVF. The appearance of QRS complex in lead III is dependent on the orientation of the QRS vector which affects the axis. There can be distorted QRS complexes frequently seen occurring in this lead because it is significantly affected by variation in breathing patterns (Figure 15).

FIGURE 15- ECG SHOWING NOTCHING OF QRS COMPLEXES IN RELATION TO RESPIRATORY VARIATION



The following table shows the different characteristic patterns of component waves of the QRS complex in the limb leads (Table 7):

TABLE 7

<table>
<tr><td>WAVE</td><td>CHARACTERISTIC PATTERN IN LIMB LEADS</td></tr>
<tr><td>Q wave</td><td>• Vector away from positively charged electrode
• Vertical axis → Q waves in II, III,aVF[23,24]
• Horizontal axis → Q waves in I,aVL
• Duration – 0.03 seconds
• Amplitude - <0.4 mV (except lead III – 0.5 mV)[18,19,24]</td></tr>
<tr><td>R WAVE</td><td>• Greatest amplitude seen in the lead which shows most parallelism and polarity between axis and maximum vector
<table>
<tr><td>Lead</td><td>Upper limit of R wave amplitude</td></tr>
<tr><td>Lead I</td><td>1.5 mV</td></tr>
<tr><td>Lead aVL</td><td>1.0 mV</td></tr>
<tr><td>Leads II,III,Avf</td><td>1.9 mV</td></tr>
</table></td></tr>
</table>

QRS MORPHOLOGY IN PRECORDIAL LEADS

The precordial leads display QRS complexes that are indicative of the vectors oriented in the horizontal plane. The waves seen are dependent on left ventricular forces in the direction of the resultant vector. The precordial leads on the right side which are V1 and V2 show mainly S wave as the dominant deflection is negative (due to leftward direction of the ventricular force vector) and the left sided precordial leads which are V5 and V6 show positive deviations in the form of R waves[25].

A progression of the R wave amplitude in the precordial leads from V1 to V6 and a reduction in the amplitude of S wave form the right to left leads can be observed. The transitional zone is situated at the lead where upright and downward deflections of the QRS complex are equal. Most commonly , the lead V3 is the site of the transitional zone, although it can be seen in any lead between V2 and V4. In lead V1, if the ratio of R wave and S wave amplitudes is greater than one, it is said to be abnormal. An R/S ratio of one is seen in normal healthy individuals in 6.4 percent of males and 1.5 percent of females in lead V1; whereas in 25 percent males and 12 percent females in lead V2. In the left precordial leads of V5 & V6, an R:S ratio of lower than one is considered abnormal[18].

TABLE 8 – characteristic patterns of component waves of the QRS complex in the precordial leads[21]

WAVE	CHARACTERISTIC PATTERN IN PRECORDIAL LEADS
Q wave	• Commonly seen in left precordial leads than right leads • Most common – V6 • Less common – V4, V5 • Rare – V3 • Duration – less than or equal to 0.03 seconds • Amplitude - <0.2 mV
R WAVE	• Increasing pattern in amplitude seen from right to left leads • Absence of R wave in V1 • Amplitude -0.6 mV (upper limit) • Tallest in lead
S WAVE	• Deepest in right precordial leads- V2 • Amplitude decreases right to left • Amplitude – 3.0 mV • QRS amplitude < 1 mV in precordial leads is considered abnormally low.

ST Segment

It is the part of the ECG recording that extends from the termination of QRS complex to the start of T wave. It is representative of the commencement of ventricular repolarization and is generally isoelectric, though deviations less than 1 mm is also normal. J point is the confluence of the end of QRS complex and initial part of the ST segment.

T Wave

A part of ventricular repolarization is recorded on the ECG by the T wave. Generally, the normal T wave either during its ascent or descent has its peak closer towards the end, thus forming an asymmetrical T wave. The T wave has positive deflections in leads V4 to V6 & lead II and negative deflection in lead aVR normally.

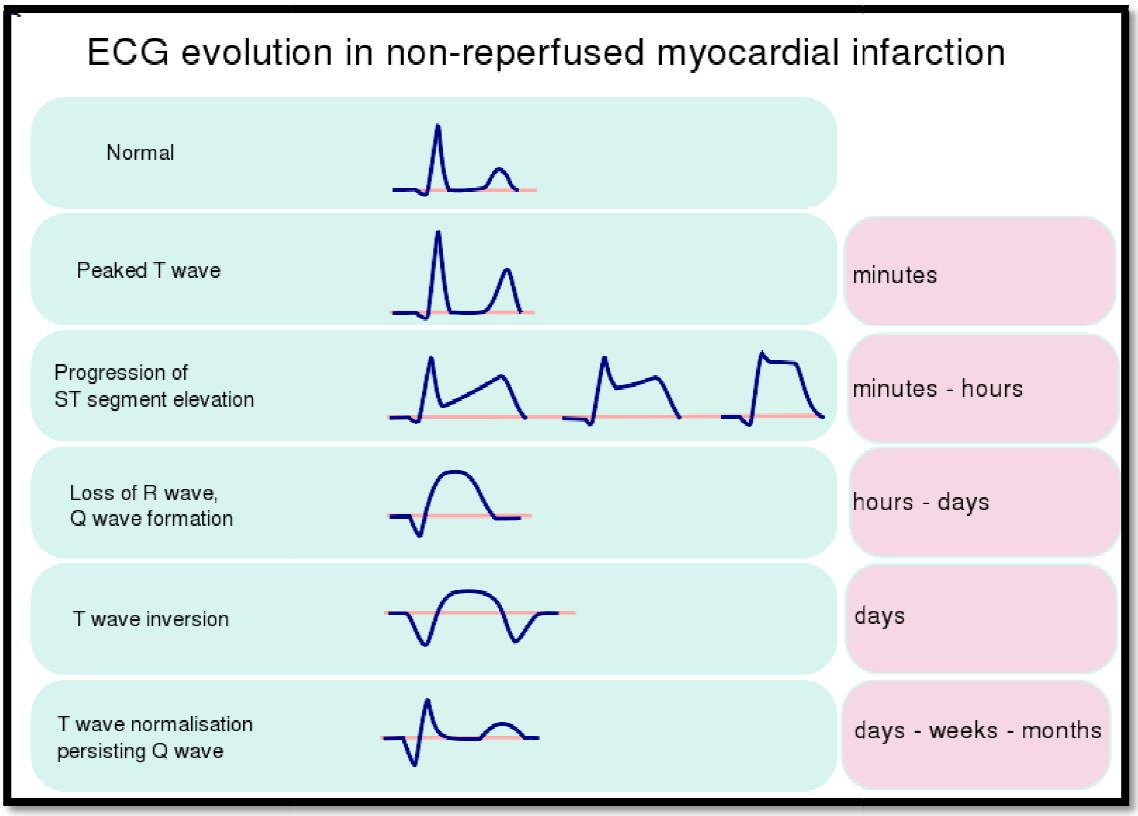
ECG CHANGES IN ST ELEVATION MYOCARDIAL INFARCTION

Ischemia and necrosis of a portion of complete thickness of left ventricular wall is called transmural MI. changes in myocardial depolarization and repolarization is generally seen in massive transmural myocardial infarctions. Sequential changes can be seen in ECG during ST elevation MI(figure 16).

In the acute phase of STEMI, due to alteration in the flow of electrical charge, there appears elevation of ST segment and occasionally positive T waves in several leads which also have similar significance as ST elevation. Simultaneously there is depression of ST segments in the reciprocal leads.

These changes are seen as early as minutes of occlusion of blood flow. In the evolving phase, (generally after few hours to days), the ST elevation begins to recede back to baseline. Simultaneously, the hyper acute J waves become inverted and progressively become deep T wave inversions[25].

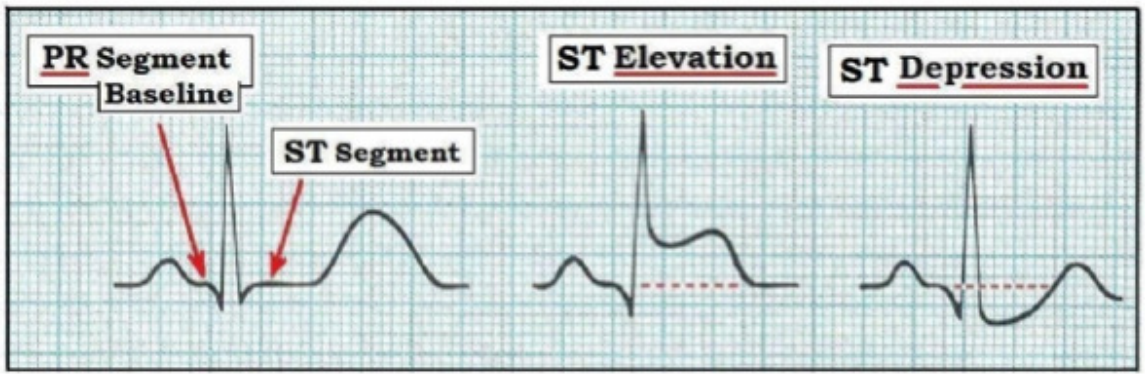
FIGURE 16



ST SEGMENT ELEVATION

In the context of an acute ischemia or infarction in a transmural MI, the first changes seen in the ECG will involve the ST segment and T wave complex in this sequence. The first of the two phases is the acute phase, in which the earliest changes are indicated by elevation of the ST segment and hyperacute T waves in some cases. This phase is what defines the STEMI. The other phase is the evolving phase, in which the sequential changes involving T waves occur causing deep inversions the same leads that showed the ST segment elevation (Figure 17). This usually happens hours to days after the onset.

FIGURE 17 – ECG SHOWING ST CHANGES IN MI



The area infarcted is known by its representative leads in the ECG. For instance, in case of anterior wall infarction, the ST-T changes will be evident on the anterior leads, namely lead I, aVL and V1 to V6, whereas if the inferior wall is involved, the sequential ECG changes of STEMI

will be seen in the leads II, III and aVF. This helps in localizing the area infarcted. The absolute explanation for ST elevation is not yet completely known. Physiologically, the normal ST segment is not positive or negative, it is iso-electric. The alteration of the ST segment reflected in the ECG is due abnormal current of injury flowing through it. Owing to infarction due to ischemia, the outer most layer of the cardiac tissue called epicardium is injured and this is the cause for the STEMI[25].

RECIPROCAL CHANGES

The phenomenon of reciprocity is a vital part of the sequential changes seen in ECG in a STEMI. However it should be borne in mind that it is not universally present in all cases. Reciprocity means that the inferior and anterior ECG leads display inversely proportional changes(Table 9). So, in case of an inferior wall STEMI, the inferior leads II, III & aVF will show ST elevation with reciprocal changes in the form of ST segment depression in anterior leads and vice versa in an anterior wall infarction(Figure 18).

The potential prognostic significance between reciprocal changes and outcome in STEMI has been under study and debate over the recent years. A study which observed the possible association between

reciprocity, left ventricular function and localization of the occluded coronary vessel compared the left ventricular function outcome in those with and without the presence of reciprocal changes in a total of 300 patients. Conclusions from this study stated that those patients presenting with anterior wall STEMI sans reciprocal changes were observed to have comparatively intact left ventricular function than those with reciprocal changes[26].

In addition to left ventricular function, reciprocity was also found to potentially localize the site of blockage of coronary vessel. In patients whose ECGs showed the phenomenon of reciprocity in apical & lateral leads, there was more incidence of multiple vessel involvement and prominent left ventricular dysfunction. The ECGs showing reciprocity in the leads I & aVL were indicative of possible localisation to RCA territory. In the study, for left ventricular dysfunction to be called significant, it had to be less than forty percent as evidenced on echocardiogram. The reason given in the literature for their conclusion is that anterior leads are mirror image reflections of apico-lateral leads. If reciprocity is observed in lateral leads it is indicative of worsening of the infarct in terms of extension of area infarcted, thereby causing more hemodynamic instability and deterioration of left ventricular function[26–28].

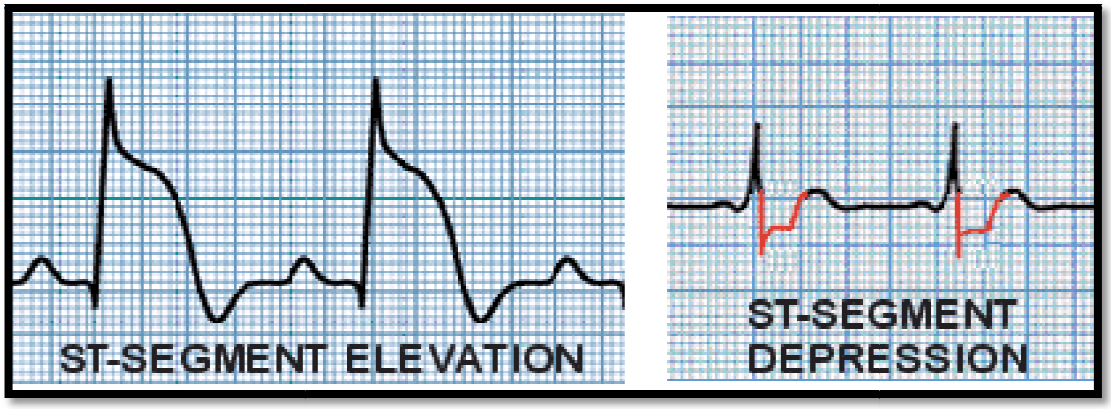
Another study done in 35 patients with STEMI with reciprocal changes, observed the association between reciprocity and need for myocardial salvage with interventional procedures for revascularization, which was assessed in terms of salvage index and area at risk (AAR). This study noted that in patients whose ECGs showed STEMI with reciprocal changes, there was a risk of greater AAR compared to those without reciprocity. This was interpreted as an indicator of bigger portion of myocardial tissue at risk of infarction, thereby necessitating an aggressive approach to revascularization in such cases[29].

TABLE 9

Overview of Infarcts

Location of Infarct	Arterial Supply	Indicative Changes	Reciprocal Changes
Anterior	LAD	**V1-V4**	II, III, aVF
Inferior	RCA	**II, III, aVF**	I, aVL
Lateral	Circumflex	I, aVL, V5, V6	V1
Posterior	Posterior Descending (RCA)	None	V1, V2
Septal	Septal Perforating (LAD) Posterior Descending (RCA	Loss of R wave in V1, V2, or V3	None

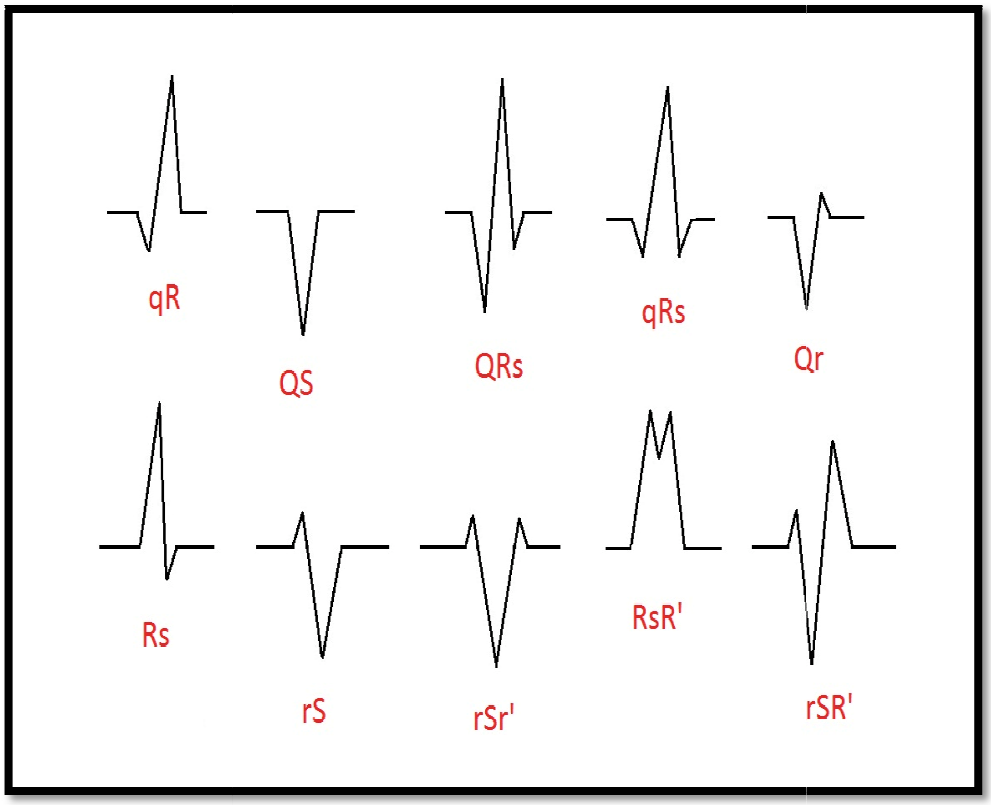
FIGURE 18 – ECG SHOWING RECIPROCAL CHANGES



EFFECT OF MYOCARDIAL INJURY ON QRS COMPLEX

The presence of myocardial tissue injury is seen in the QRS complex as loss of positive amplitude in the ECG lead representative of the affected area causing the appearance of Qr, QS complex or loss of R wave voltage[25](Figure 19).

FIGURE 19 - SHOWING VARIOUS DEFLECTIONS AND COMPLEXES OF THE QRS



QS complex

- As the name suggests, there is no positive deflection in this type of QRS complex. It is a representation of total absence of QRS upward deflection(positivity).

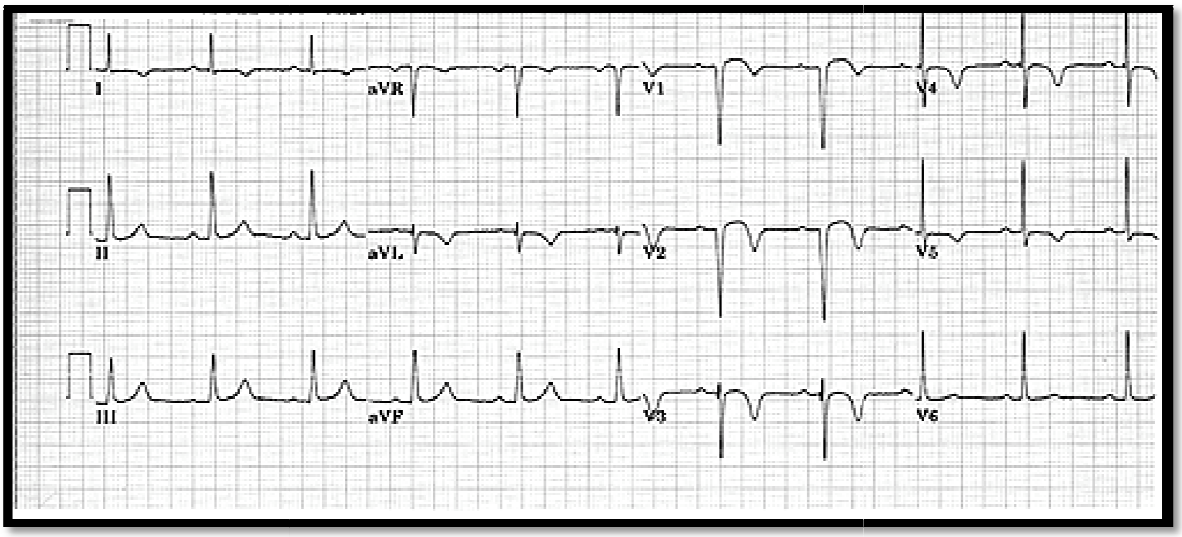
- When myocardial necrosis occurs, the tissue becomes electrically dead with no potential for depolarization. If a transmural infarct occurs, the whole thickness of muscle becomes electrically inert leading to the formation of a hole in the myocardial wall.

- As the hole is electrically neutral, it will show the same activity as the nearest normal tissue as reflected through the window. This is the rationale behind the orientation of the vector representative of depolarization of infarcted ventricle.

- The vector that is supposed to reflect the activity in the free ventricular wall will instead represent the depolarisation of the septum causing a downward (negative) deflection as it is oriented away from the electrode. The subsequent vector representative of the right ventricular wall will also show a negative deflection for the same reason (figure 20).

- The QS complex is a reflection of the vectorial law of ECG in MI which states that " QRS vectors are directed away from infarcted myocardium"[25].

FIGURE 20 – ECG SHOWING ANTERIOR WALL INFARCTION WITH QS COMPLEXES



Qr complex

- As represented in the name and size of letters, the Qr complex constitutes an initial large abnormal Q wave which is wide and deep succeeded by a comparatively smaller r wave which is the positive upward deflection(Figure 21).

- This complex occurs when the infarction is not totally transmural and also has patchy involvement of the tissue above (epicardium) or below (endocardium) the myocardium.

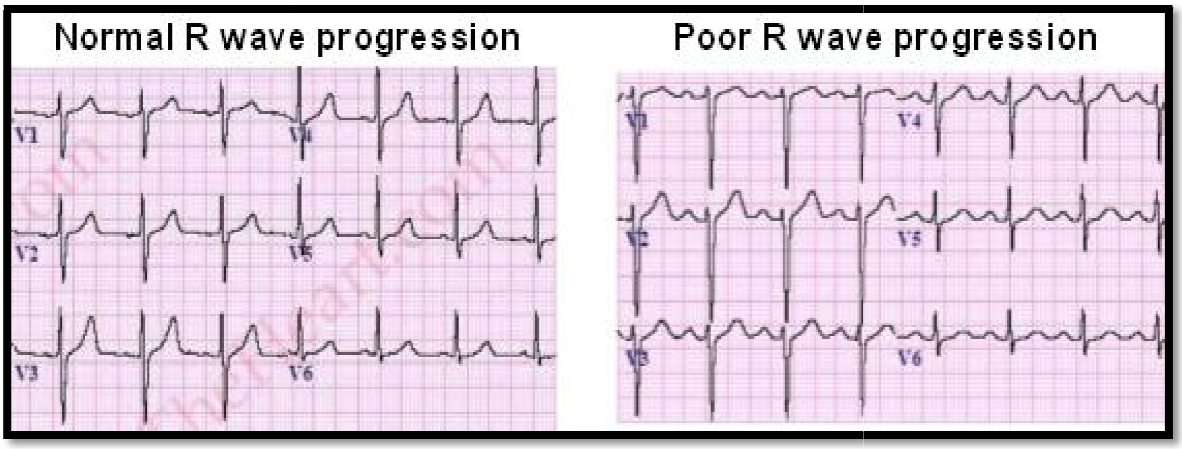
- Each of the deflection has a significance in relation to this type of infarct. The first pathological Q wave is a reflection of the infarcted tissue causing loss of QRS positivity. The subsequent r wave is a manifestation of depolarization of residual living cardiac tissue overlying the infarcted area.

FIGURE 21 – ECG SHOWING QR complex

Loss of the R wave amplitude

- If an ECG lead is said to have loss of R wave amplitude if there is diminished R wave voltage without an abnormal Q wave(Figure 22).
- This phenomenon is supposed to occur in the leads placed in the boundary of the area of ischemia.
- In the setting of a myocardial infarction, this finding is often ignored probably because of excessive focus on finding the Q wave[25].
- The mechanism behind loss of R wave amplitude is similar to that of full thickness infarcts causing the QRS being oriented away from the electrode and loss of positivity.
- The presence of epicardial or endocardial focal infarcts causes decreased extent of the QRS vector poiting towards the electrode. So abnormal Q waves don't occur as the diminished QRS vectors are enough to counterbalance the QRS vectors oriented away from the area of infarct.

FIGURE 22- showing comparison of normal R wave progression and loss of R wave amplitude after infarction



ECG as a prognostic marker in myocardial infarction

There have been various studies regarding the usefulness of the ECG as a prognostic marker in MI. Data from the GUSTO-I database (which studied forty one thousand and twenty one patients), from which a multi-variate analysis was done suggests a correlation between certain ECG characteristics and clinical outcome, which was evaluated in terms of mortality at thirty days of infarction [30](Table 10).

TABLE 10 - showing the ECG variables predictive of 30 day mortality according to a multi-variate analysis of the GUSTO-I database[30].

ECG variable	*Characteristic predictive of thirty day mortality*	*Odds ratio*
Heart rate (beats per minute)	More than 84	2.47
Sum total of ST elevation and depression	More than or equal to 19 mm	1.53
QRS complex width in case of anterior wall infarcts	Duration more than or equal to 100 milli seconds	1.55
In case of inferior infarct	ECG showing signs of previous infarct in acute inferior infarction	2.47

In addition to the above mentioned parameters, an additional two variables observed to be predictive of a poorer prognosis are the occurrence of Q waves of new onset and absence of resolving pattern in the ST-T wave changes after the infarction.

Two of the above mentioned ECG parameters, sum total of ST deviation (elevation and depression) and signs of older infarction in acute

inferior MI were found to be at least partially, directly indicative of the degree of damage to the myocardial tissue.

Further trials were done to substantiate this evidence, like the GISSI-1 study, and stated that the ST segment deviation sum total is indeed a strong predictor of mortality. This observation was not only seen at one month post-infarction, but also proved consistent after six months of the primary event[31]. In addition to these observations, it was also noted that those patients with greater areas of infarction had therapeutic advantage with revascularization after thrombolysis[32].

In terms of area of the infarction, when anterior and inferior wall myocardial infarctions were compared, the patients with anterior wall STEMI appeared to have a poorer prognosis than those with inferior wall STEMI[33–37].

The explanation given in the literature of the study was that anterior infarcts were more likely to have a larger area of ischemic damage. Another study stated that this difference in outcome was due to the higher incidence of complications in anterior infarcts like worse left ventricular function, occurrence of arrhythmias and heart failure[34]

ECHOCARDIOGRAM (ECHO)

The most comprehensive and widely used heart imaging tool is Echocardiography. Cardiac valvular function, morphology of cardiac chambers (size & shape), real time assessment of hemodynamics inside the heart and diastolic & systolic functions can be evaluated during echocardiography. Echocardiography has advantage of being low in cost and non invasive nature resulting in minimal patient inconvenience[14]. Echo is based on principles of Ultrasound.

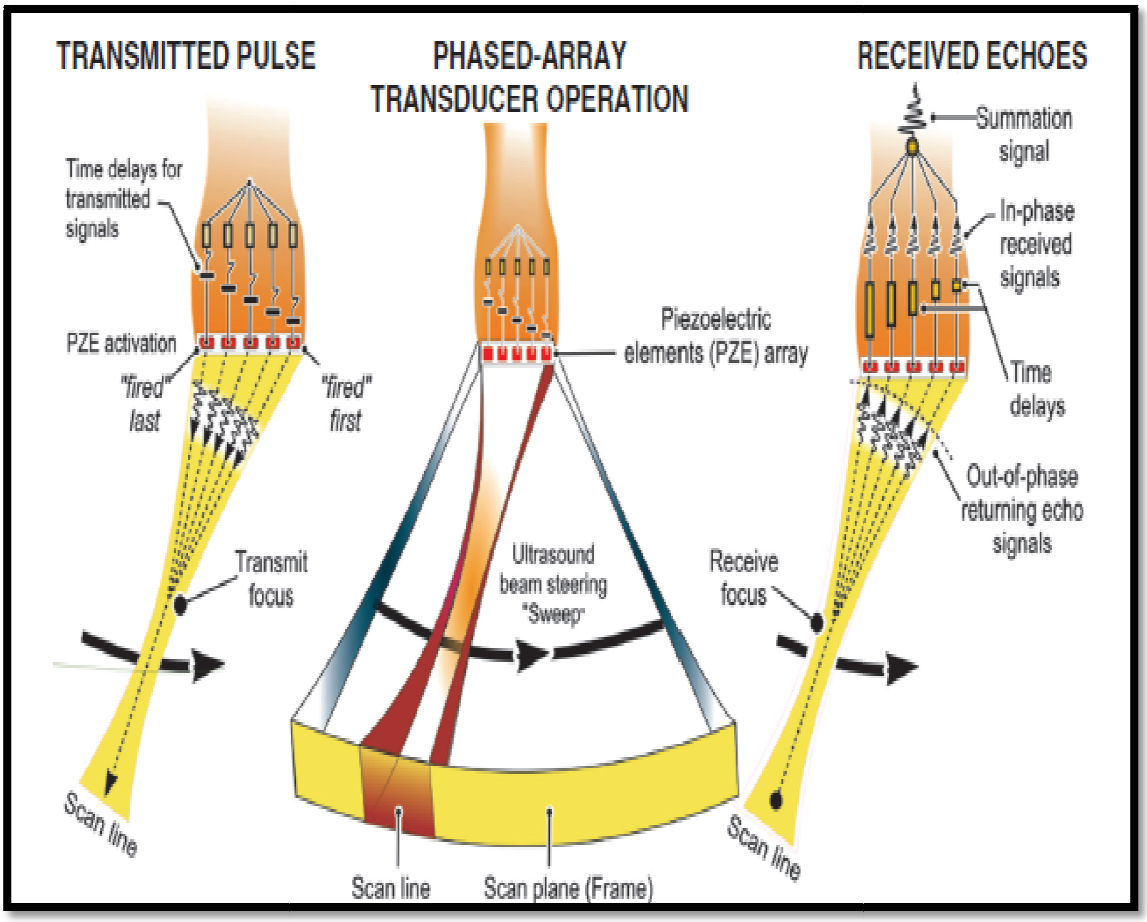
IMAGE GENERATION

The basic principles of ultrasound imaging are which echocardiography also works on. The transducer consists of piezoelectric crystals which generate sound waves of high frequency type (1to 10 Mhz) which interact with tissues and return back to transducer after passing through many structures inside the body, then the ultrasound machines compute the reflecting structures depth based on time taken by the waves to travel to and fro.

M – MODE (motion mode) – wherein the singular scan line (from singular ultrasound beam) recorded on a moving paper/screen plots time and depth on horizontal and vertical axis respectively. Though becoming obsolete this mode is thought to be best suited for linear measurements assessment and wherein temporal resolution is high[14].

TWO DIMENSIONAL MODE – wherein the phased array transducers which are electronically directed and work based on pulse echo principle(Figure 23). The ultrasound pulses are repeatedly produced and echoes recorded in an sequential and orderly manner to produce moving images. The evaluated tissue depth & restricted ultrasound speed regulate the pulse repetition frequency(PRF).

FIGURE 23



DOPPLER IMAGING

Cardiac chamber movement quantification and the flow of blood (i.e velocity) through various chambers and major vessels can be done based on Doppler principle.

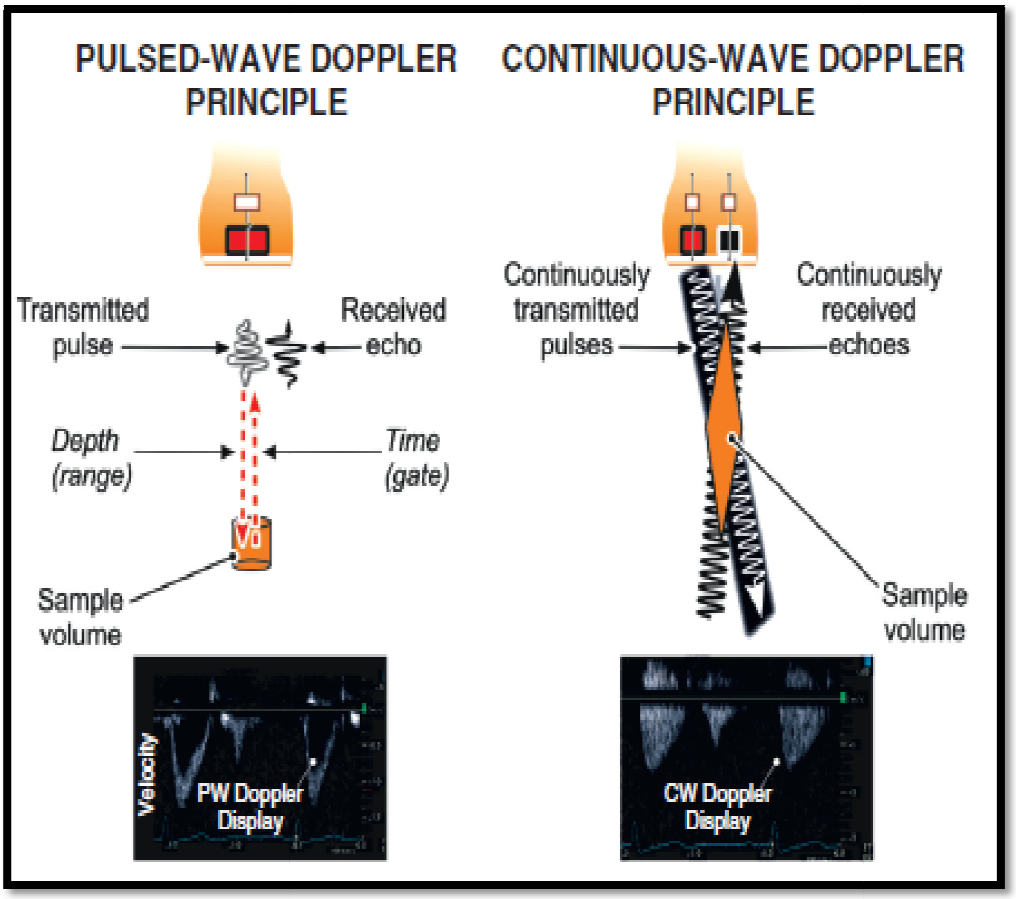
Doppler Principle: Frequency of waveform emitted from a moving object will be discerned as greater or less than the actual frequency based on movement of object to or away from the observer.

CONTINUOUS & PULSE WAVE DOPPLER

These are two main varieties of Doppler Imaging. Ultrasound waves are sent out continuously and received by separate piezoelectric elements in Continuous Wave Doppler (CW Doppler) (Figure 24). In CW Doppler depth analysis is not feasible and but no restriction on velocities being measured.

Ultrasound waves are sent at discrete intervals which are reflected off moving objects and recorded by the transducer in Pulse Wave Doppler (PW Doppler) (Figure 24). In PW Doppler, gating of Ultrasound waves helps in measuring velocity (<1.5m/sec) at a certain depth.

FIGURE 24



COLOUR FLOW DOPPLER

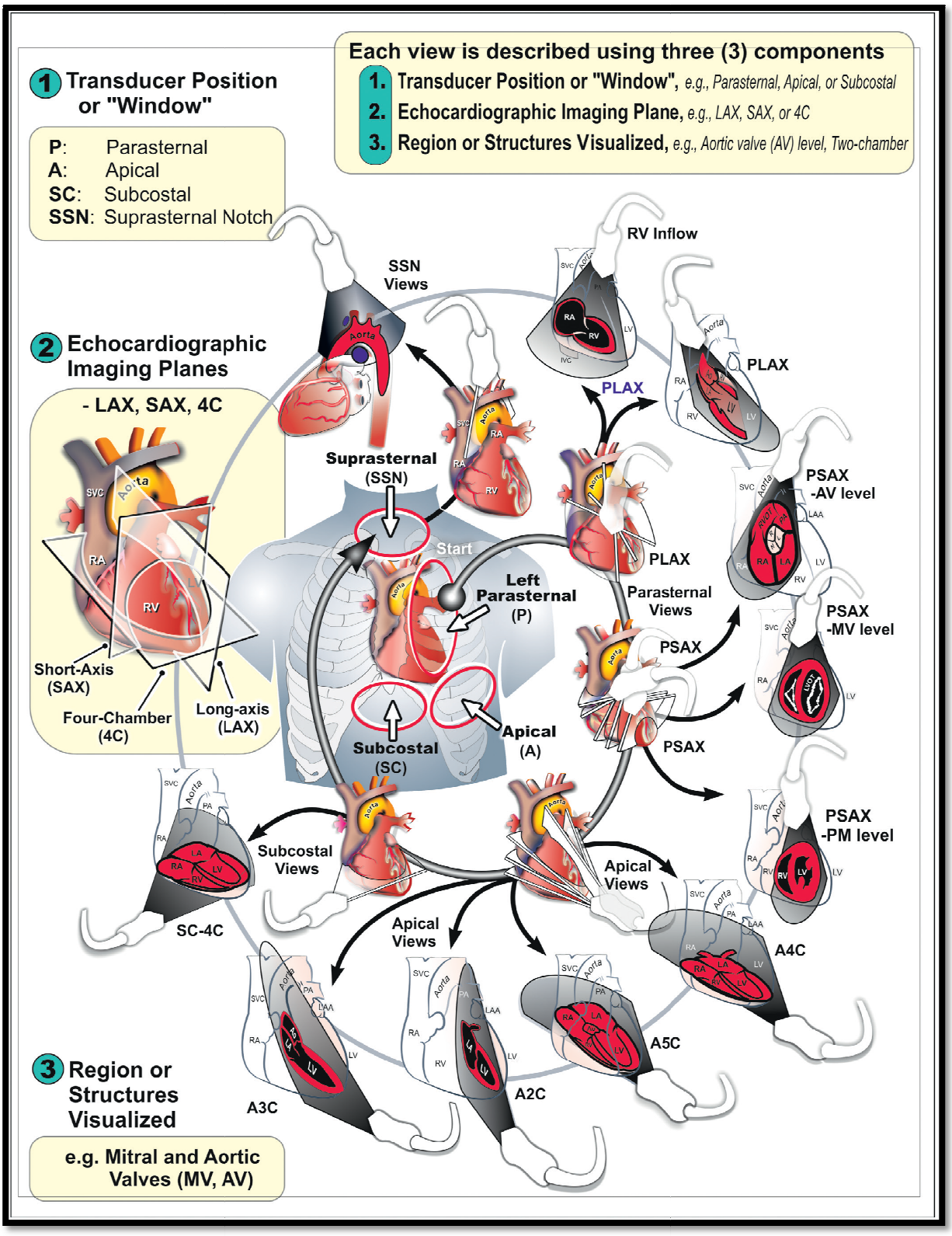
It is pulse wave Doppler based technique wherein flow towards the transducer and away are encoded in red & blue respectively in the interested area. Mosaic & multicoloured pattern (yellow & green usually) are seen in case where flow is turbulent and of high varying velocities.

STANDARD ADULT TRANSTHORACIC ECHO EXAMINATION

Doppler imaging, two dimensional and M-mode combination constitute standard adult TTE. In the protocol recommended for complete examination, includes best image acquisition wherein 3 main components are used to describe echo views (Figure 25).

1. The standard transducer window
2. The orthogonal echo imaging planes
3. The interested anatomical region.

The following figure (figure 25) shows the different echocardiographic imaging planes:

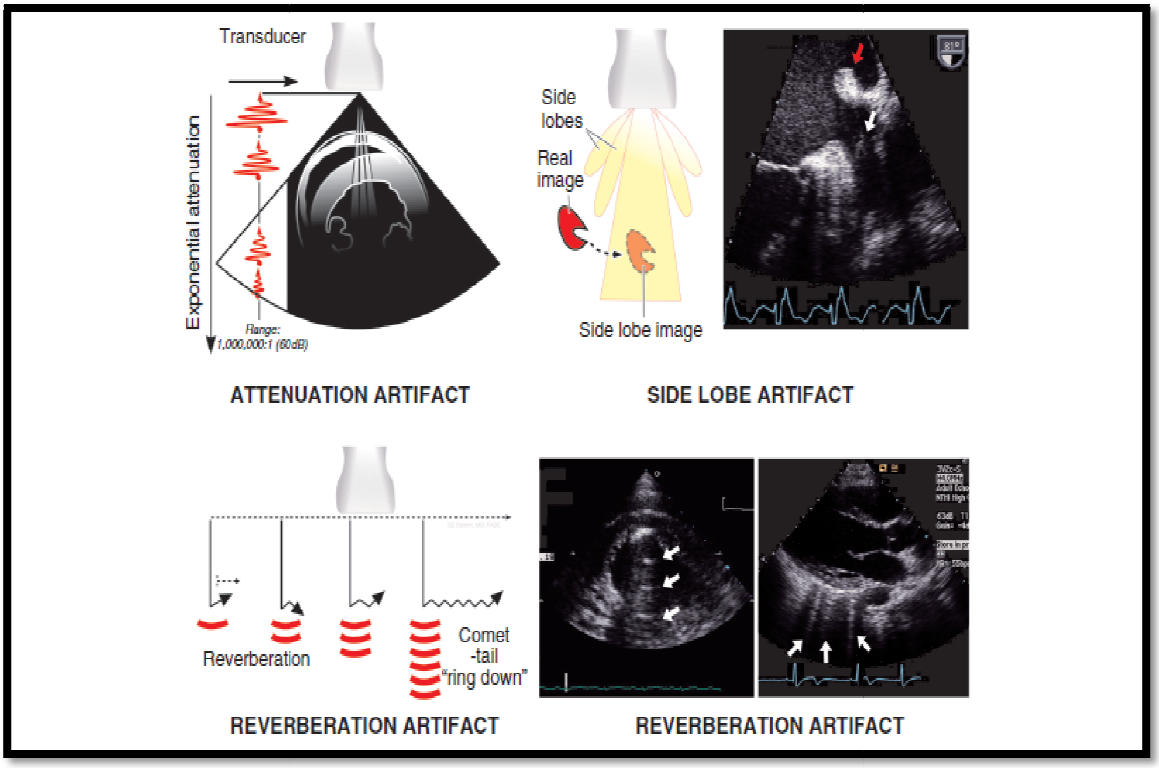


M-Mode ECHO : Left ventricular chamber size on parasternal view, posterior & septal wall thickness are measured. Other uses are diastolic function measurement in Colour M-Mode.

IMAGING ARTIFACTS

Due to ultrasound physical fundamentals result in majority chunk of imaging artifacts which are quite common in echo(Figure 26).

FIGURE 26



LEFT VENTRICULAR ASSESSMENT

STRUCTURE: SIZE & MASS

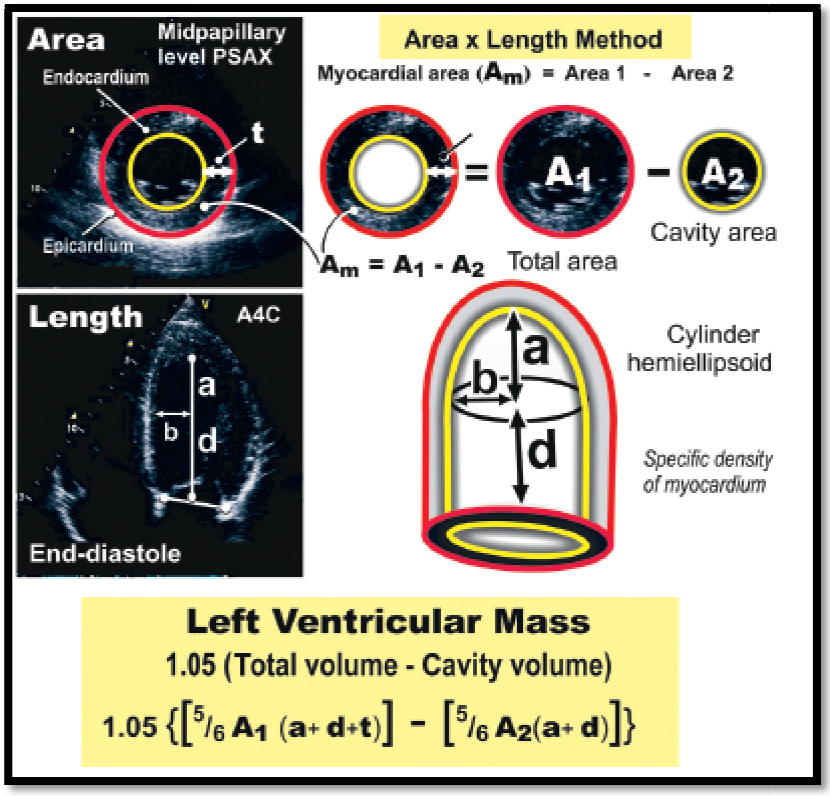
One of the demonstrated, accurate and favoured method for volume assessment independent of rigid geometric assumptions is the single / biplane Simpson method[14]. In this method, in four and/ or two chamber views after recognising the endocardial border with the

assistance of the computer, ventricular diameter is measured from which cross sectional area is calculated.

The limitations of this method are difficulty of endocardial border identification when image quality is poor, errors in volumetric estimation due to minor alteration in transducer angle leading to fore-shortening of ventricle.

Chamber size and wall thickness are used in several formulae to assess left ventricular mass though their correctness is diminished when geometry of ventricle is altered (Figure 27).

FIGURE 27 – SHOWING ESTIMATION OF LV MASS USING AREA LENGTH METHOD IN 2D ECHO



LEFT VENTRICULAR SYSTOLIC FUNCTION

The ejection fraction of the LV can be devised by substracting the end systolic volume from the end diastolic volume and then dividing it by end diastolic volume. It is one of the most widely used methods for detection and risk assessment of many cardiovascular pathologies.

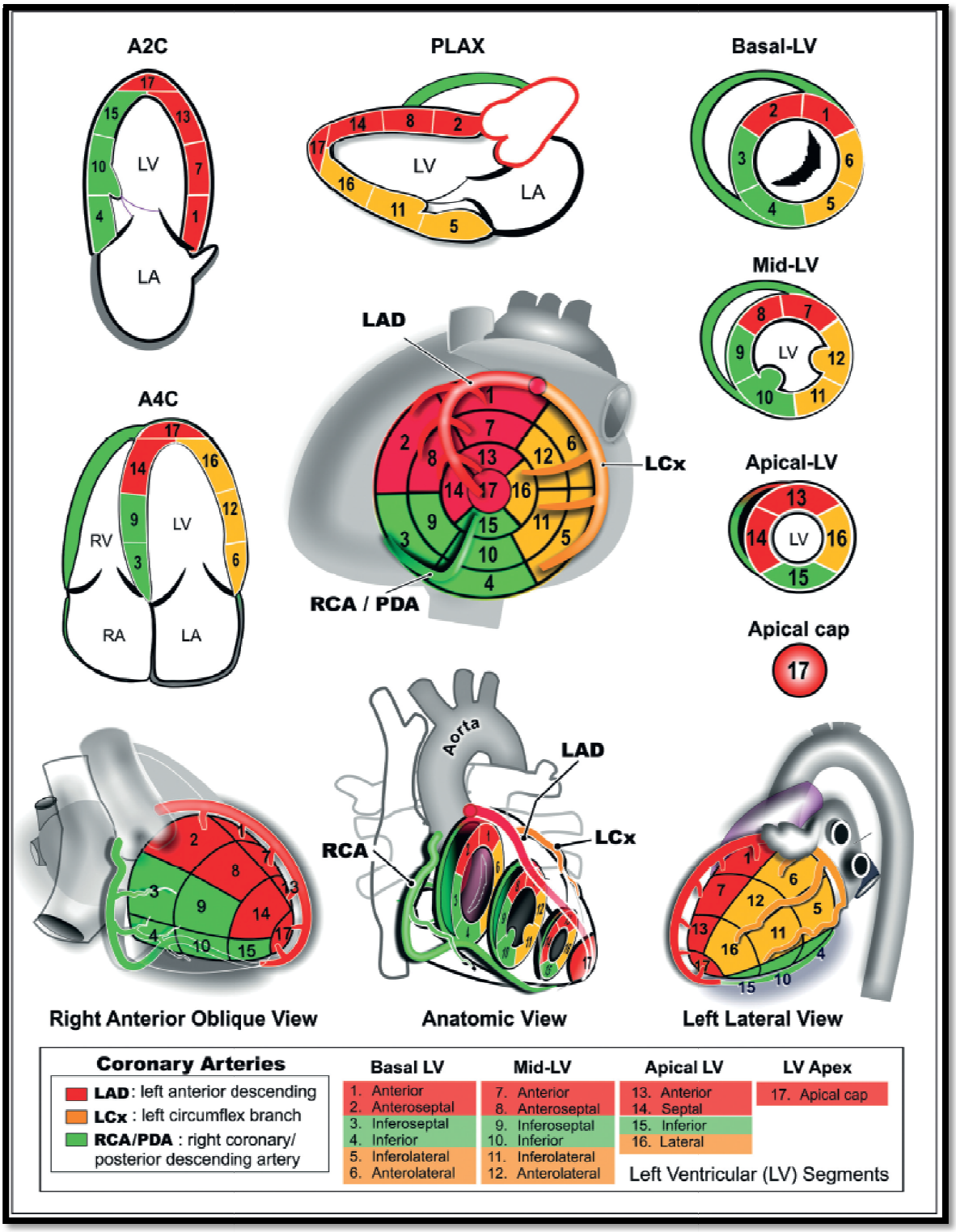
Though LV ejection fraction calculation by volumetric method is ideal, due to multiple variables visual method is more accurate in seasoned echocardiographer. Myocardial strain imaging is a new way of estimating function of the heart and can also be performed using Doppler imaging.

REGIONAL FUNCTION OF THE LEFT VENTRICLE

When a particular coronary blood vessel is affected in acute myocardial infarction , regional wall motion abnormalities according to its individual area of distribution.

Scoring systems have been developed to calculate regional wall motion abnormality, of which the most extensively practiced method was devised by the American society of echocardiography which is based on the seventeen segment model (Figure 28).

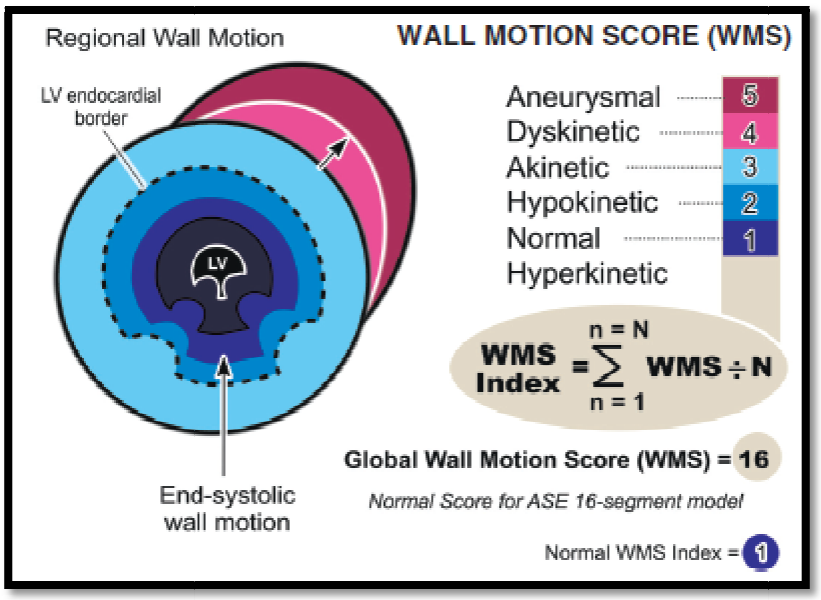
FIGURE 28 - SHOWING SEVENTEEN SEGMENTS OF LEFT VENTRICLE -



In this method, LV segments are scored as(Figure 29) :

- dyskinetic – 4 points
- akinetic – 3 points
- hypokinetic – 2 points
- normal – 1 point

FIGURE 29 - SHOWING WALL MOTION SCORE INDEX



$$\text{Wall motion score index (WMSI)} = \frac{\text{Sum of these grades}}{\text{Number of segments visualized}}$$

WMSI = 1.0 (normokinetic ventricle)

WMSI $\geq$ 1.7 (physical examination findings of heart failure)

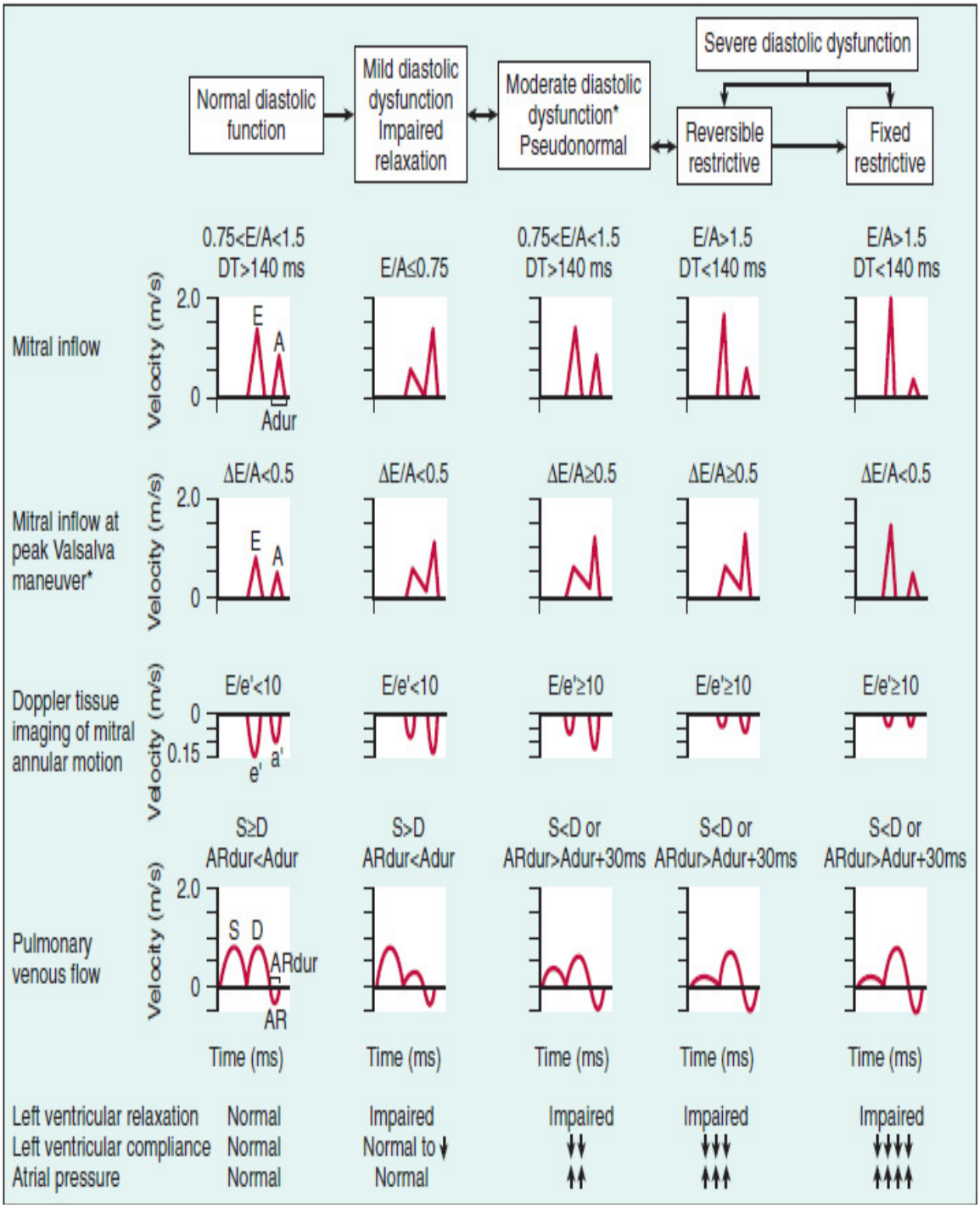
In the scenario of heart failure following MI , a higher WMSI score is a reliable indicator of worse morbidity and mortality[38]. The prime aim of recognizing RWMA is detection of patients suffering from coronary artery disease. But echo cannot differentiate acute & previously existing regional wall motion anomalies.

In the scenario of new onset myocardial infarction RWMA can be picked within minutes of ischemic injury, this is especially useful in situations where there is a suspicion of acute coronary syndrome with ambiguous ECG findings. Differential diagnosis for the presence of RWMA can be sarcoidosis & myocarditis. But a clear pattern of coronary hypoperfusion is unlikely in such cases.

LEFT VENTRICULAR DIASTOLIC FUNCTION

The estimation of left ventricular diastolic function by non invasive means is controversial because the gold standard method is the invasive pressure volume loop assessment. Owing to its good resolution echo with doppler is ideal for detection of diastolic dysfunction(figure 30).

FIGURE 30- showing diastolic function abnormalities as seen in echo colour Doppler.



A= Transmitral flow velocity with atrial contraction

a`= velocity of mitral annulus motion with atrial systole

Adur= duration of A

AR flow from left atrium to pulmonary veinsduring atrial contraction

ARdur= duration of AR

D = diastolic

DT= decceleration time

E= early diastolic transmitral flow velocity

e`= velocity of early diastolic mitral annular motion

S= systolic

In routine clinical practice the evaluation of diastolic function needs numerous data from Doppler imaging, Mitral inflow patterns & Pulmonary venous patterns(figure 30). In asymptomatic patients diastolic function can be evaluated using exercise testing which may unveil diastolic dysfunction causing exertional symptoms[14].

RESULTS

A total of 120 patients were included in the study. Six patients were excluded because the area of STEMI was inferior wall and posterior wall. The extent of infarct would have been a confounding factor had they been studied alongside those with inferior infarcts alone. So a total of 114 patients presenting with ST elevation myocardial infarction were included.

Demographic details of the study:

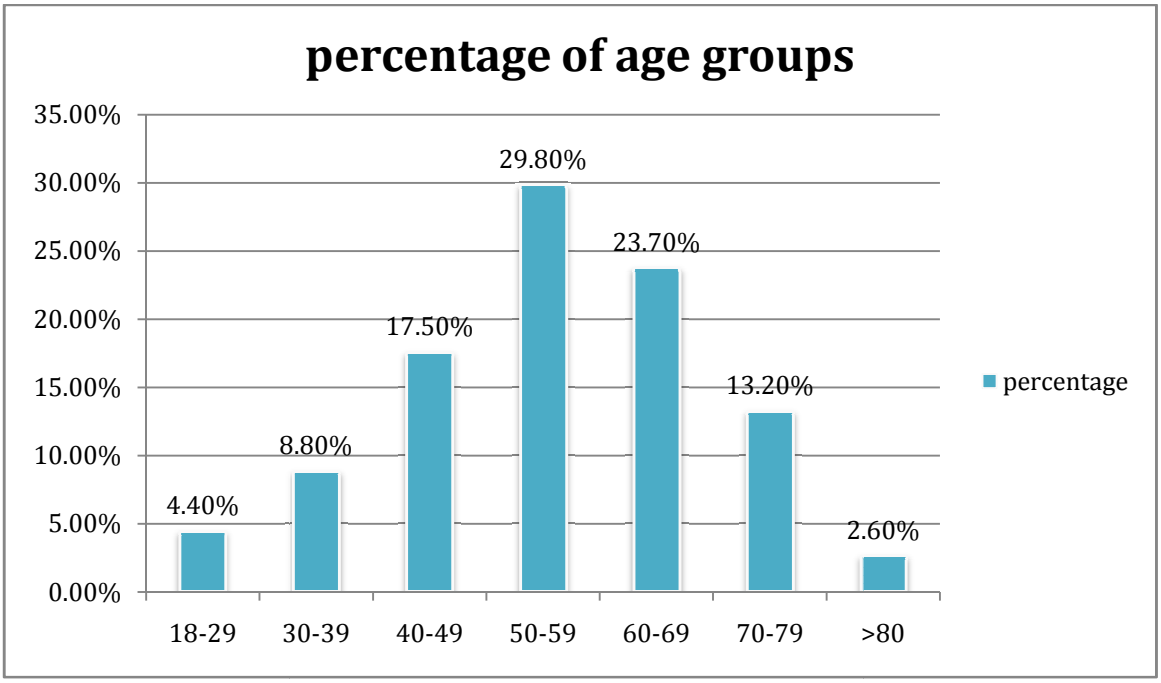
Of the 114 patients included in the study, 95 patients were male and 19 patients were female.

Among the 114 patients, youngest patient was 26 years old and the oldest patient was 81 years old. Majority of the patients (29.8%) were in the 50 – 59 years age group(figure 31 & table 11).

TABLE 11

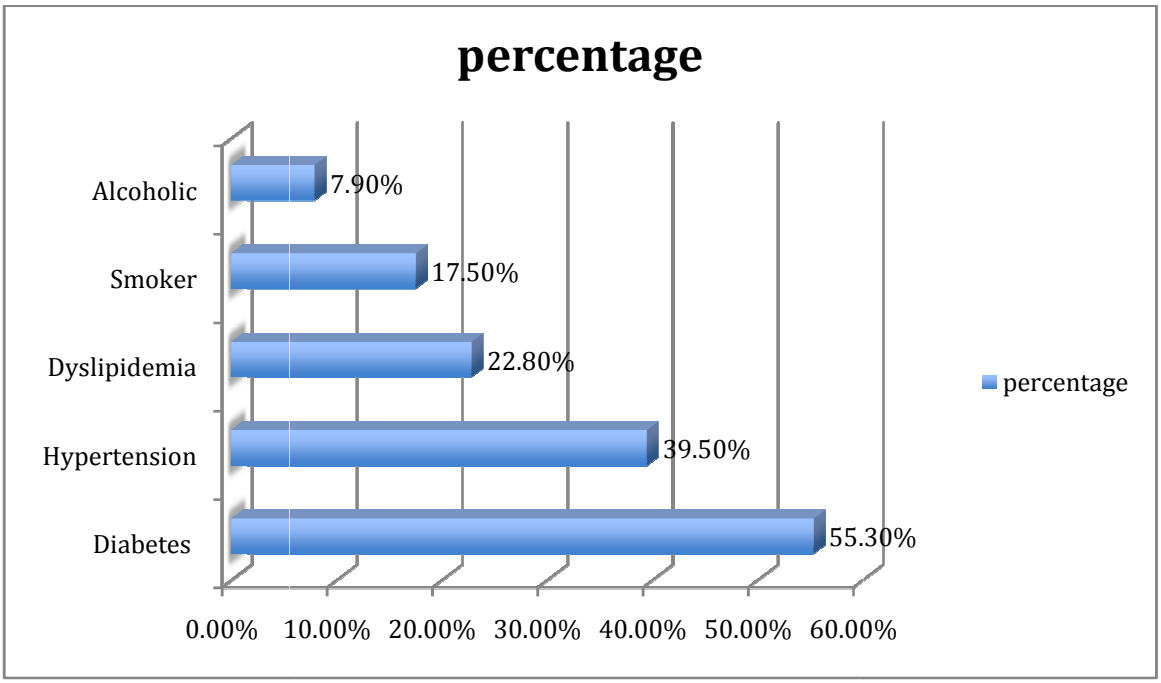
Age group (in years)	Number of patients (n= 114)	Percentage
18-29	5	4.4%
30-39	10	8.8%
40-49	20	17.5%
50-59	34	29.8%
60-69	27	23.7%
70-79	15	13.2%
>80	3	2.6%

FIGURE 31



Among the study population, 63 (55.3%) patients had diabetes, 45 (39.5%) were hypertensive and 26 (22.8%) had dyslipidemia. There were 20 (17.5%) patients were smokers and 9 (7.9%) patients were known alcoholics. (shown in figure 32)

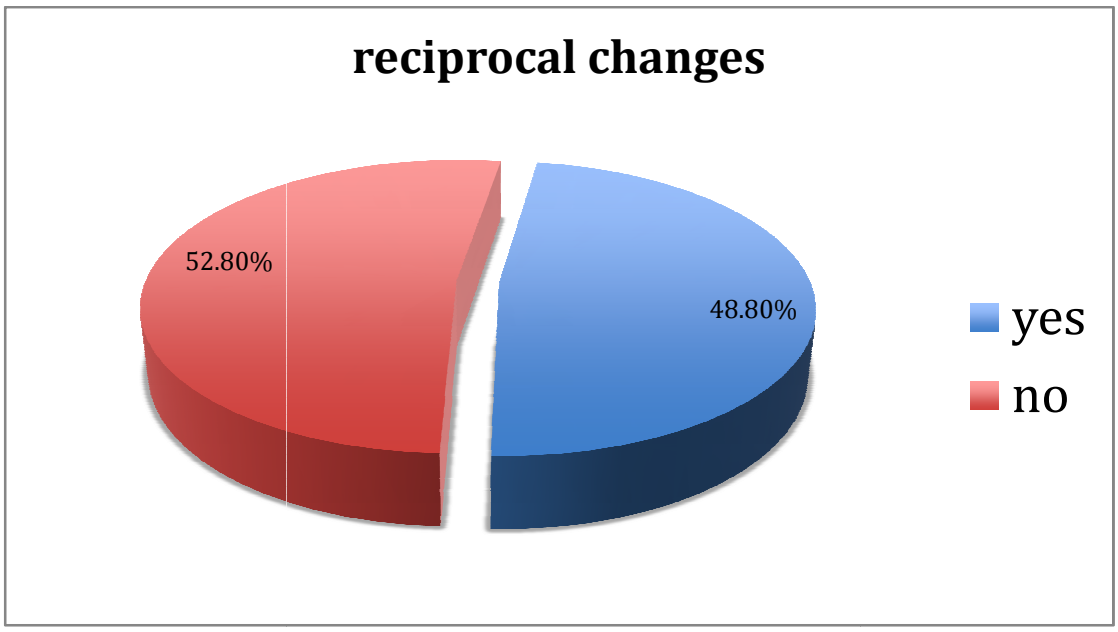
FIGURE 32



Out of the total 114 patients studied, 78 (68.4%) had anterior wall STEMI and 36 (31.6%) had inferior wall STEMI.

Among the total study population, 55 (48.2%) patients had reciprocal changes in ECG (Figure 33).

FIGURE 33

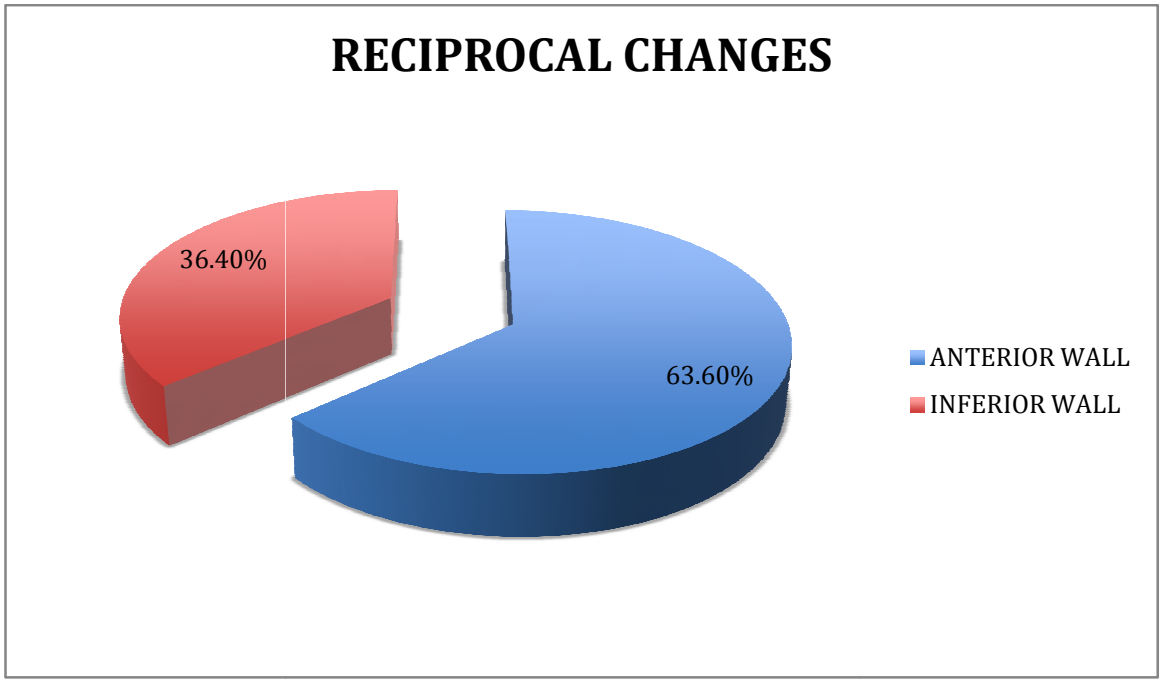


Of the 78 patients with anterior wall STEMI, 35 (63.6%) had reciprocal changes in ECG. Of the 36 patients with inferior wall STEMI, 20 (36.4%) had reciprocal changes (Table 12 & Figure 34).

TABLE 12

RECIPROCAL CHANGES	ANTERIOR WALL STEMI (n = 78)	INFERIOR WALL STEMI (n = 36)
YES	35 63.6%	20 36.4%
NO	43 72.9%	16 27.1%

FIGURE 34

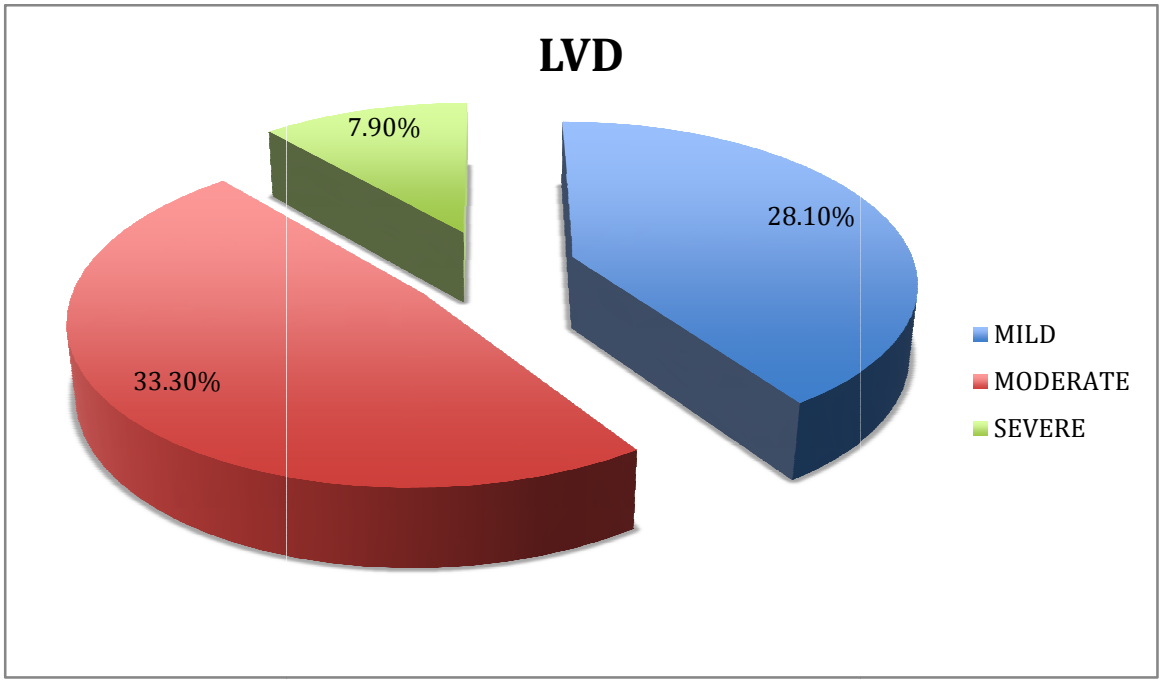


In the 114 STEMI patients studied, 35 patients (30.7%) had adequate left ventricular function and the remaining 79 patients (69.3%) had left ventricular dysfunction. Out of the 79 patients with left ventricular dysfunction, 32 patients (28.1%) had mild LVD, 38 (33.3%) patients had moderate LVD and 9 patients (7.9%) had severe LVD (Table 13 & Figure 35) .

TABLE 13

LEFT VENTRICULAR FUNCTION	NUMBER OF PATIENTS	PERCENTAGE
Adequate LV function	35	30.7%
Mild LVD	32	28.1%
Moderate LVD	38	33.3%
Severe LVD	9	7.9%

FIGURE 35



Out of 114 patients admitted with STEMI, 39 (34.2%) patients were treated with medical management alone, 27 (23.7%) patients underwent thrombolysis, 7 (6.1%) patients underwent percutaneous intervention (PCI), 33 (28.9%) patients underwent PTCA, 5 (4.4%) patients underwent thrombolysis along with PTCA and 3 patients (2.6%) underwent CABG.

Among the 114 STEMI patients studied, 106 patients came for follow up and 8 patients were lost to follow up. Out of 106 patients, 72(63.2%) were categorized as NYHA class I based on symptoms at first follow up. 29(25.4%) were graded as class II and 5(4.4%) patients as class III based on NYHA classification(Figure 36 & Table 14). There were no patients with class IV symptoms on first follow up.

FIGURE 36

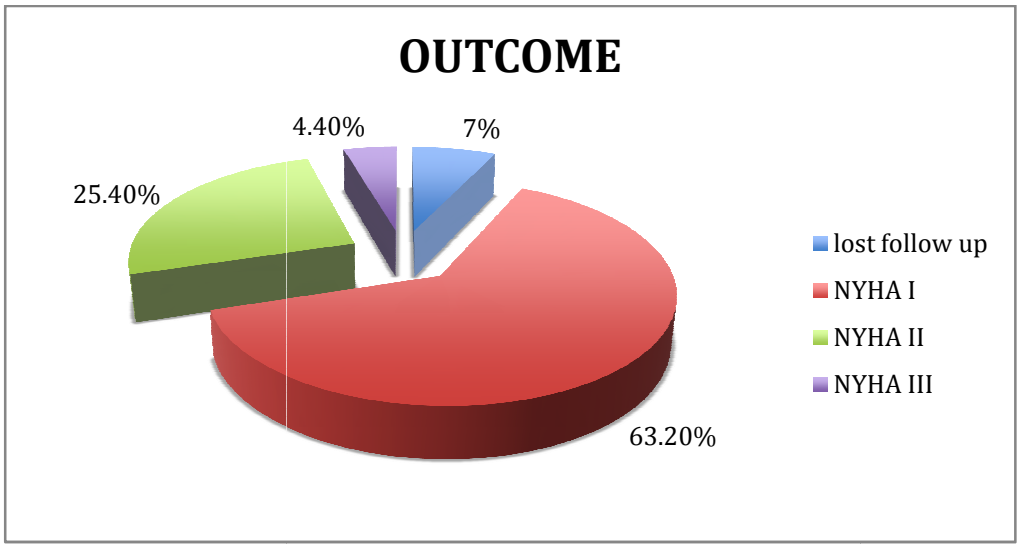


TABLE 14

Area of STEMI	Lost to follow up	NYHA I	NYHA II	NYHA III	TOTAL
AWMI	7 9%	48 61.5%	19 24.4%	4 5.1%	78 100%
IWMI	1 2.8%	24 66.7%	10 27.8%	1 2.8%	36 100%
TOTAL	8 7%	72 63.2%	29 25.4%	5 4.4%	114 100%

Out of 55 patients with reciprocal changes, 38 patients (69.1%) developed left ventricular dysfunction(Table 15). The association between the two variables was found significant (chi square test value = 0.002).

TABLE 15

RECIPROCAL CHANGES	**LVD**		**Pearson Chi square p value**
	YES	**NO**	
YES	38 69.1%	17 30.9%	0.002
NO	41 69.5%	18 30.5%	

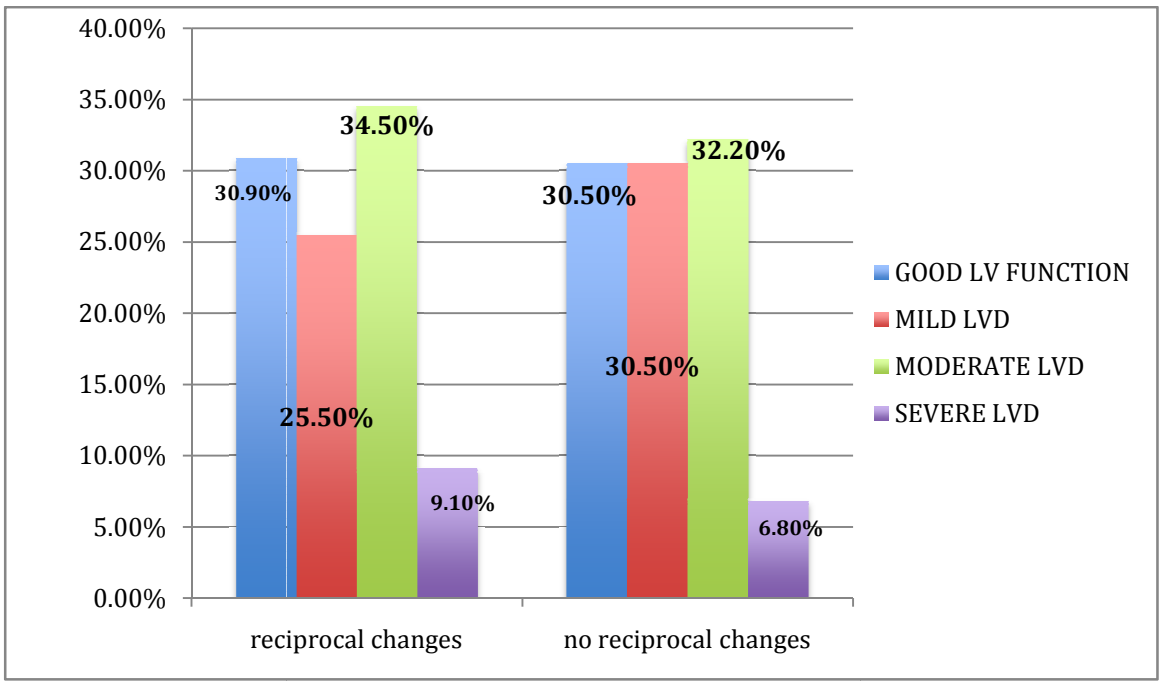
Of the 55 patients who had reciprocal changes in their ECGs, 17(30.9%) had adequate left ventricular function, 14 (25.5%) patients had mild LVD, 19 (34.5%) had moderate LVD and the remaining 5 (9.1%)patients had severe LVD (Table 16).

The association between the two variables was found to be statistically insignificant (p value = 0.919).

TABLE 16

RECIPROCAL CHANGES	LVD			Pearson Chi square test value	P value
	MILD	MODERATE	SEVERE		
YES	14 25.5%	19 34.5%	5 9.1%	0.500	0.919
NO	18 30.5%	19 32.2%	4 6.8%		

FIGURE 37



In the 78 patients with anterior wall STEMI, 35 patients had reciprocal changes(Table 17 & Figure 38). The association between presence of reciprocal changes and left ventricular dysfunction was found using chi square test and was insignificant (p value = 0.843).

Out of the 35 patients with reciprocal changes, 6 patients(40%) had adequate left ventricular function (no LVD) and out of the 43 patients without reciprocal changes, 9 patients (60%) had adequate left ventricular function (no LVD). (Table 17 & Figure 38)

FIGURE - 38

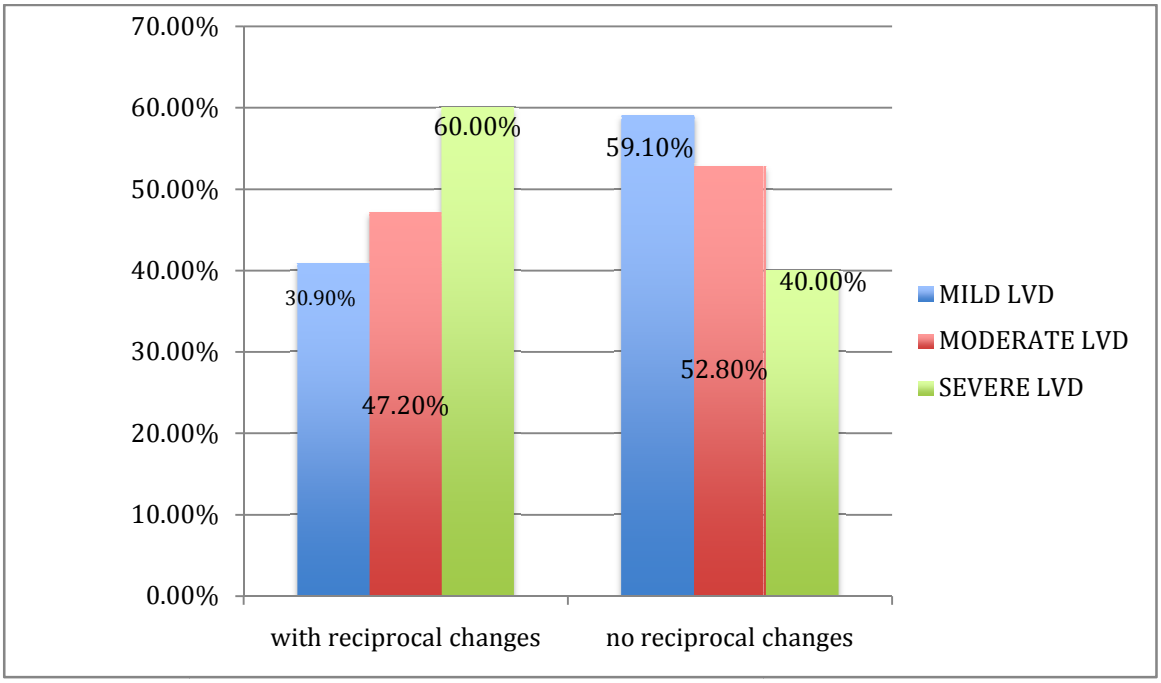

70.00%
60.00%
50.00%
40.00%
30.00%
20.00%
10.00%
0.00%
30.90%
47.20%
60.00%
59.10%
52.80%
40.00%
with reciprocal changes
no reciprocal changes
MILD LVD
MODERATE LVD
SEVERE LVD

TABLE – 17

LVD	RECIPROCAL CHANGES		TOTAL
	NO	YES	
NO LVD	9 60%	6 40 %	15 100%
MILD LVD	13 59.1%	9 40.9%	22 100%
MODERATE LVD	19 52.8%	17 47.2%	36 100%
SEVERE LVD	2 40%	3 60%	5 100%
TOTAL	43 53.1%	35 44.9%	78 100%

In the 36 patients with inferior wall STEMI, 20 patients had reciprocal changes.(Table 18) The association between presence of reciprocal changes and left ventricular dysfunction was found using chi square test and was insignificant (p value = 0.856).

Out of these 20 patients with reciprocal changes, 11 patients(55%) had adequate left ventricular function (no LVD) and out of

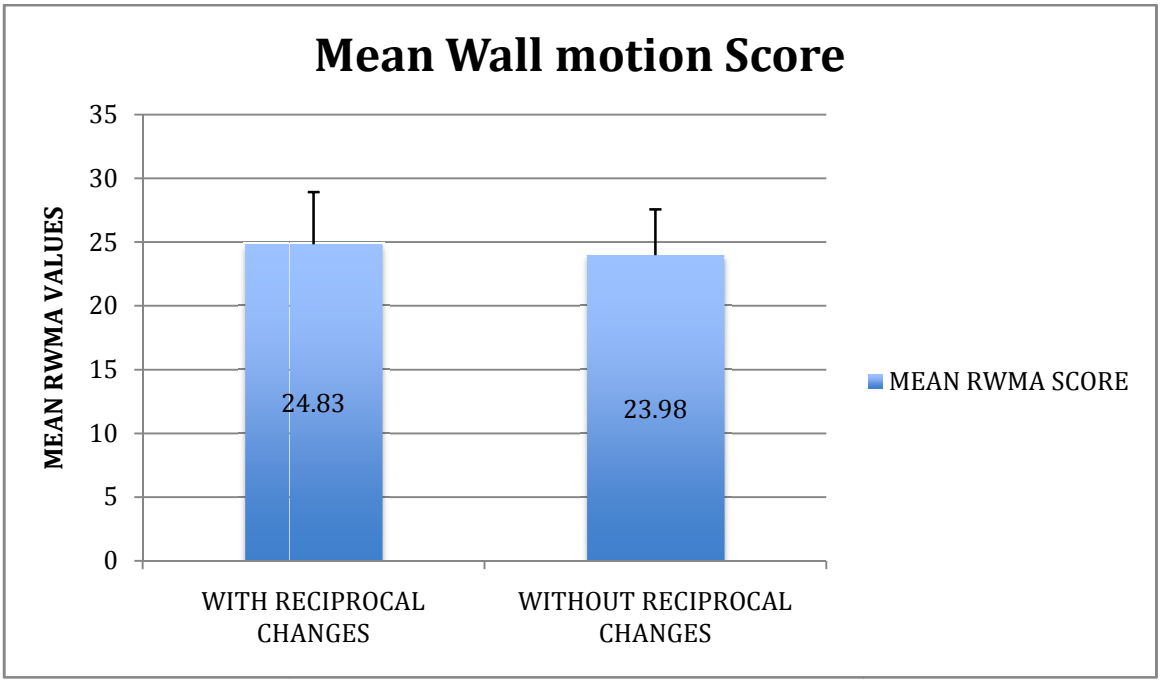
the 16 patients without reciprocal changes, 9 patients (56.3%) had adequate left ventricular function (no LVD). (Table 18)

TABLE - 18

RECIPROCAL CHANGES	NO LVD	MILD LVD	MODERATE LVD	SEVERE LVD	TOTAL
NO	9 56.3%	5 31.3%	0 0%	2 12.5%	16 100%
YES	11 55%	5 25%	2 10%	2 10%	20 100%
TOTAL	20 55.6%	10 27.8%	2 5.6%	4 11.1%	36 100%

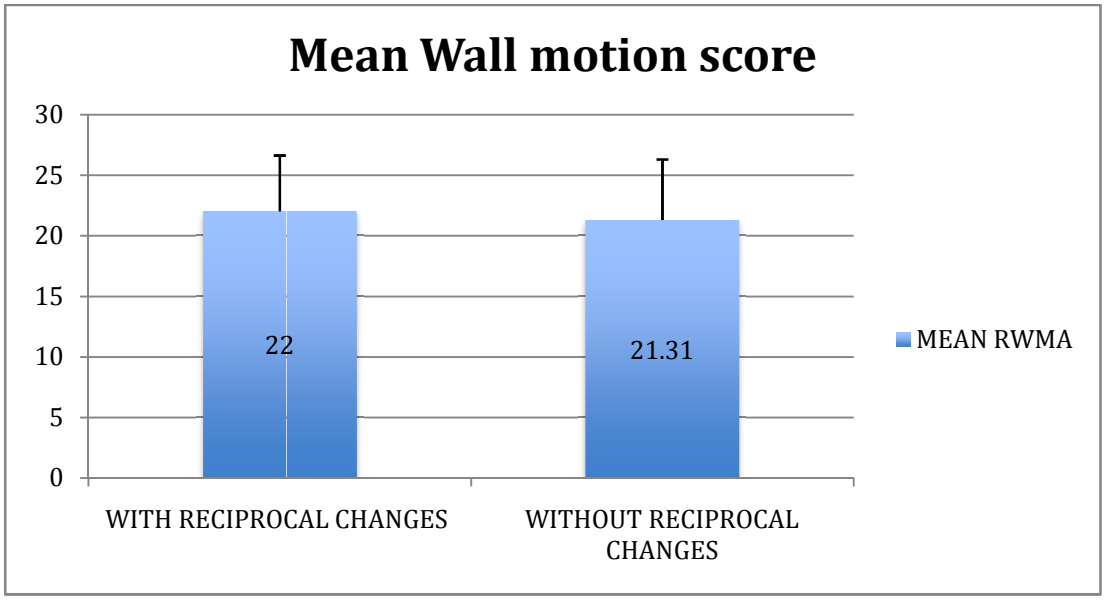
In the 78 patients with anterior wall infarction, the association between reciprocal changes and Wall motion score was analysed. The mean Wall motion score in patients with reciprocal changes was 24.83 ± 4.1. The mean Wall motion score in patients without reciprocal changes was 23.98 ± 3.6. The difference between the two groups was not significant (p value = 0.342). (Figure 39)

FIGURE 39



In the 36 patients with inferior wall infarction, the association between reciprocal changes and Wall motion score was analysed. The mean Wall motion score in patients with reciprocal changes was 22 ± 4.623. The mean Wall motion score in patients without reciprocal changes was 21.31 ± 4.99. The difference between the two groups was not significant (p value = 0.431). (Figure 40)

FIGURE 40



The mean QRS amplitude score in all patients with left ventricular dysfunction was 33.25 ± 16.34. The association was analysed using ANOVA test and was found to be statistically insignificant (p value 0.089).

(Table 19)

The association between QRS amplitude score and left ventricular dysfunction was analyzed in patients with anterior wall STEMI using ANOVA test and was found to be statistically insignificant (p value 0.307). (Table 19)

The association between QRS amplitude score and left ventricular dysfunction was analyzed in patients with inferior wall STEMI using ANOVA test and was found to be statistically insignificant (p value 0.156). (Table 19)

TABLE 19

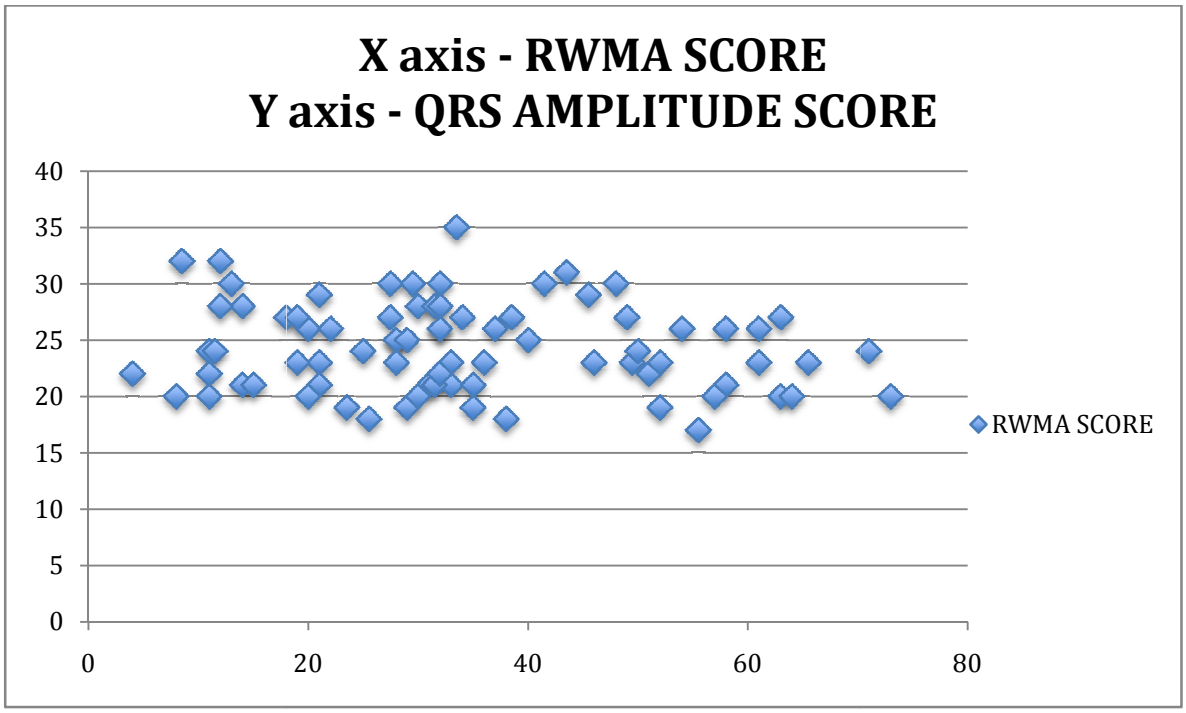
Analysis of QRS AMPLITUDE SCORE vs LVD	**ANOVA value**	**P value**
In all patients (n = 114)	2.23	0.089
Anterior wall STEMI patients (n = 78)	1.225	0.307
Inferior wall STEMI patients (n = 36)	1.863	0.156

The mean QRS amplitude in all the 114 patients was 34.17 ± 16.929. The mean Wall motion score in all 114 patients was 24.36 ± 3.915. Using pearson chi square test, the correlation was statistically significant.

(r value = **- 0.210**)

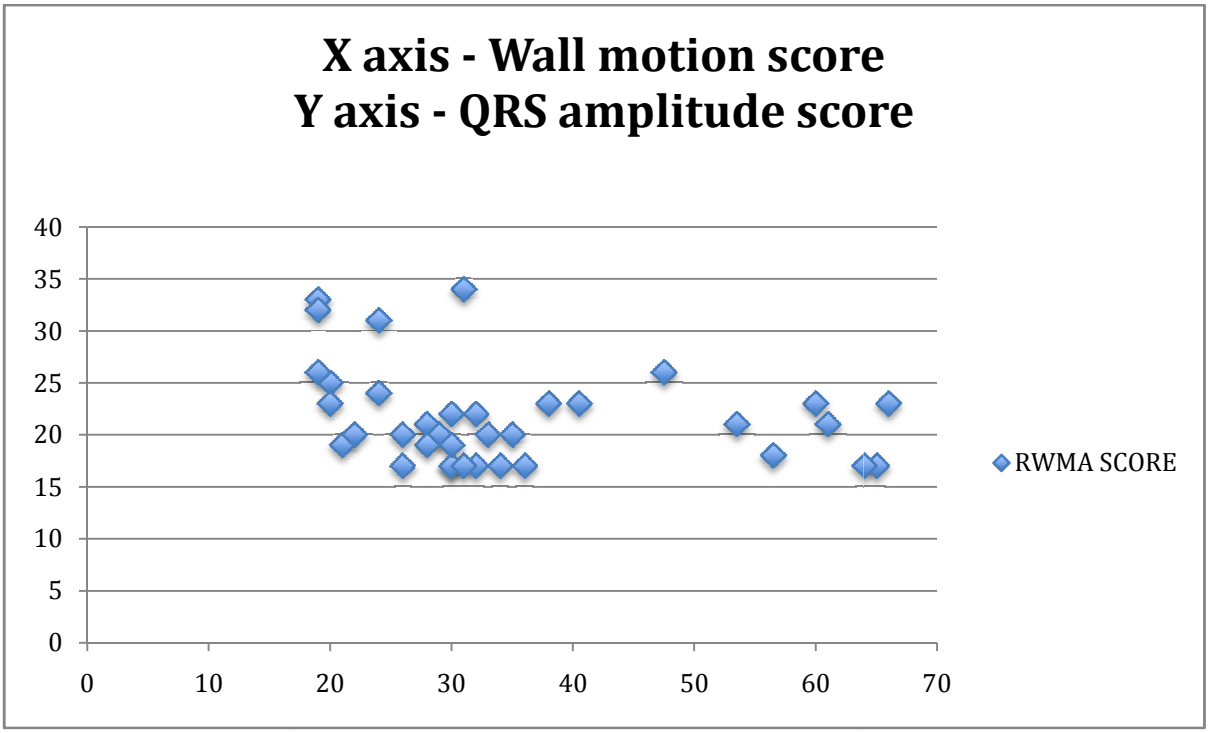
The mean QRS amplitude score in anterior wall infarction patients was 34.17 ± 16.9. The mean Wall motion score in anterior wall infarction patients was 24.36 ± 3.9. Using the pearson chi square test, the correlation was found to be statistically significant (r value = **- 0.147**). (Figure 41)

FIGURE- 41



The mean QRS amplitude score in inferior wall infarction patients was 35.03 ± 14.4. The mean Wall motion score in anterior wall infarction patients was 21.69 ± 4.7. Using the pearson chi square test, the correlation was found to be statistically significant (r value = **- 0.359**) (Figure 42)

FIGURE- 42



Out of the 78 patients with anterior wall infarction, 35 patients had reciprocal changes in their ECGs. Among those 35 patients, 15 (42.9%) had no symptoms NYHA class I, 14 (40%) patients had NYHA class II symptoms, 4 (11.4%) patients had NYHA class III symptoms and 2(5.7%) were lost to follow up. The p value calculated using Pearson Chi square test and was found to be statistically significant($p = 0.001$). (Table 20)

TABLE - 20

RECIPROCAL CHANGES	OUTCOME AT FIRST FOLLOW UP			
	NYHA I	NYHA II	NYHA III	LOST TO FOLLOW UP
YES	9 45%	10 50%	1 5%	0 0%
NO	15 93.8%	0 0%	0 0%	1 6.3%

Out of the 36 patients with inferior wall infarction, 20 patients had reciprocal changes in their ECGs. Among those 20 patients, 9 (45%) had no symptoms NYHA class I, 10 (50%) patients had NYHA class II symptoms and 1 (5%) patient had NYHA class III symptoms. The p value calculated using Pearson Chi square test was statistically significant($p = 0.004$).

(Table 21)

TABLE 21

RECIPROCAL CHANGES	OUTCOME AT FIRST FOLLOW UP			
	NYHA I	NYHA II	NYHA III	LOST TO FOLLOW UP
YES	15 42.9%	14 40%	4 11.4%	2 5.7%
NO	33 76.7%	5 11.6%	0 0%	5 11.6%

Among the 78 patients with anterior wall STEMI, 48 patients had no symptoms at first follow up (NYHA I) and 23 patients were symptomatic (NYHA II and above). In these 48 patients under NYHA class I, the mean QRS amplitude score was 33.95 ± 18.17. In these 23 patients under NYHA class II and above, the mean QRS amplitude score was 35.30 ± 15.12. The difference between the two groups was statistically insignificant($p = 0.759$).

Among the 36 patients with anterior wall STEMI, 24 patients had no symptoms at first follow up (NYHA I) and 11 patients were symptomatic (NYHA II and above). In these 24 patients under NYHA class I, the mean QRS amplitude score was 34.70 ± 13.47. In these 11

patients under NYHA class II and above, the mean QRS amplitude score was 36.72 ± 17.17. The difference between the two groups was statistically insignificant (p = 0.706).

Among the 78 patients with anterior wall STEMI, 7 patients (9%) were lost to follow up. Among the 36 patients with inferior wall STEMI, 1 patient (2.8%) was lost to follow up. All of these 8 patients were excluded from the analysis.

DISCUSSION

ST elevation myocardial infarction is one of the most widely seen cause for hospital admission and mortality. The age at which a person encounters an infarction has been steadily decreasing with more patients presenting with myocardial infarction from younger age groups due to various causative factors. When the frequency distribution of age was analysed in our study, it showed 4.4% of the patients were in the 18-29 age group. The majority of the patients were in 50 – 59 years age group.

In concordance with established studies, majority of the patients (95 of 114 patients) were males. Diabetes appeared to be the leading risk factor for ST elevation MI with 55.3% of the study population being diabetic followed by hypertension. The predominance of diabetes in STEMI patients is consistent with the findings in a study by Mcmanus et al which observed recent trends in STEMI[39].

In our study, more than two- thirds of the patients had anterior wall infarction and the remaining had inferior wall infarction. Reciprocal changes in the ECG were seen in about half of the patients (48.2%) of which around two thirds had anterior wall infarcts and the remaining had inferior wall infarcts.

Of the patients studied, majority (69.3%) had left ventricular dysfunction following STEMI. On admission, 34.2% of patients were treated with medical therapy alone whereas the remaining underwent various modes of intervention like PTCA, PCI, thrombolysis etc.

The presence of reciprocal changes in ECG showed significant association with development of left ventricular dysfunction when analysed in all the patients in total. When the subset of patients with reciprocal changes in anterior wall and inferior wall myocardial infarction were considered separately, the association with LVD was statistically insignificant.

In anterior wall MI patients, the presence of reciprocal changes did not reflect in an increase in the Wall motion score. When a similar association was analysed in inferior wall MI patients, it was also found to be insignificant.

The association between Wall motion score and QRS amplitude score was analysed. It was observed that a decrease in QRS amplitude score was associated with an increase in Wall motion score in both anterior and inferior wall myocardial infarction patients.

When the association between QRS amplitude score and degree of LVD was found, it was statistically insignificant. This means that a low QRS amplitude score does not reflect the degree of LVD.

The association between reciprocal changes and clinical outcome in terms of NYHA class of symptoms on first follow up was found to be significant. This may imply that the presence of reciprocal changes can spell worse outcome following ST elevation myocardial infarction when compared to those without reciprocal changes in their ECG.

The assessment of outcome at follow up has confounding factors like compliance with medication, diet and lifestyle modification measures. So it is a limitation of the study.

CONCLUSION

The following conclusions were made based on the findings of our study:

- ST elevation myocardial infarction was most common in the age group of 50 – 59 years and with male preponderance.
- Diabetes mellitus appeared to be the leading risk factor for STEMI.
- Majority of the patients (more than half) developed left ventricular dysfunction following myocardial infarction.
- The patients who had reciprocal changes in their ECG were more prone to developing left ventricular dysfunction. But this significance was lost when individual areas of infarction were considered.
- The presence of reciprocal changes was not associated with a higher wall motion score indicating the degree of infarction.
- The finding of low QRS amplitude in the ECG didn't directly lead to the development of left ventricular dysfunction. LVD was present irrespective of the QRS amplitude score.
- In patients with lower QRS amplitudes, higher wall motion scores were observed indicating that low voltage of QRS complexes in the ECG can be predictive of larger extent of the infarct.
- The presence of reciprocal changes in the ECG can signify poorer outcome on follow up at three to four weeks after infarction.

- Low QRS amplitude is not predictive of the clinical outcome following STEMI.

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PROFORMA

1.NAME

2.AGE

3.SEX

4.CO-MORBIDITIES:

- Diabetes/Hypertension/Dyslipidemia
- Smoking/Alcohol
- Others

5.TYPE OF TREATMENT:

Medical/intervention(thrombolyis/PTCA/PCI/CABG)

6.ECG AT ADMISSION:

- RECIPROCAL CHANGES – present/absent
- QRS AMPLITUDE SCORE – sum of R wave amplitudes of all leads in the ECG

7.TWO DIMENSIONAL ECHOCARDIOGRAM:

- WALL MOTION SCORE – seventeen segments of LV
- EJECTION FRACTION – eyeballing method

8.CLINICAL OUTCOME: At first follow up after discharge

- NYHA class

ABBREVIATIONS

ECG	–	Electrocardiogram
LVD	–	left ventricular dysfunction
ASE	–	American society of echocardiography
NYHA	–	New York Heart Association
SPSS	–	Statistical Package for the Social Sciences
ANOVA	–	Analysis of Variance
STEMI	–	ST Elevation Myocardial Infarction
MI	–	Myocardial Infarction
S1	–	first heart sound
S2	–	second heart sound
EF	–	Ejection Fraction
CI	–	Cardiac index
PCWP	–	Pulmonary Capillary Wedge Pressure
ANS	–	Autonomic Nervous System
VF	–	Ventricular Fibrillation
LAD	–	Left Anterior Descending Artery
RCA	–	Right Coronary Artery

LCX	–	Left Circumflex Artery
Echo	–	Echocardiogram
SA node	–	Sinoatrial Node
AV	–	Atrio-Ventricular
AAR	–	Area at risk
M – Mode	–	Motion Mode
PRF	–	Pulse Repetition Frequency
CW Doppler	–	Continuous Wave Doppler
PW Doppler	–	Pulse Wave Doppler
A	–	Transmitral flow velocity with atrial contraction
a`	–	velocity of mitral annulus motion with atrial systole
Adur	–	duration of A
AR	–	flow from left atrium to pulmonary veinsduring atrial contraction
ARdur	–	duration of AR
D	–	diastolic
DT	–	decceleration time

E	–	early diastolic transmitral flow velocity
e`	–	velocity of early diastolic mitral annular motion
S	–	systolic
PTCA	–	Percutaneous Transluminal Coronary Angioplasty
PCI	–	Percutaneous Coronary Intervention

CONSENT FORM

PSG Institute of Medical Science and Research, Coimbatore

Institutional Human Ethics Committee
INFORMED CONSENT FORMAT FOR RESEARCH PROJECTS

I , DR SILPITA KATRAGADDA, am carrying out a study on the topic:
CORRELATION OF QRS AMPLITUDE & RECIPROCAL CHANGES IN ECG TO OUTCOME IN FIRST TIME ST-ELEVATION MYOCARDIAL INFARCTION
as part of my research project being carried out under the aegis of the Department of:

GENERAL MEDICINE

(*Applicable to students only*): My research guide is: DR LS SOMASUNDARAM

The justification for this study is: Considering disease burden and the morbidity associated with myocardial infarction, early prediction of outcome is essential for treatment and prevention of complications

The objectives of this study are:

Primary Objective: TO OBSERVE THE CORRELATION BETWEEN ECG AT ADMISSION & ECHO FINDINGS AFTER 48 HOURS IN PATIENTS WITH FIRST TIME ST ELEVATION MYOCARDIAL INFARCTION

Secondary Objective: TO ASSESS THE CLINICAL OUTCOME AFTER ONE MONTH OF FOLLOW UP IN PATIENTS WITH FIRST TIME ST ELEVATION MYOCARDIAL INFARCTION AND TO CORRELATE IT WITH ECG & ECHO FINDINGS DURING ADMISSION.

Sample size: All patients admitted with ST elevation MI in the study period-between January 2014 to September 2014.

Study volunteers / participants are (specify population group & age group): Patients above the of 18 years admitted with ST ELEVATION MYOCARDIAL INFARCTION.

Location: PSG HOSPITALS, COIMBATORE

We request you to kindly cooperate with us in this study. We propose collect background information and other relevant details related to this study. We will be carrying out:

Initial interview (specify approximate duration): 15 minutes.

Data collected will be stored for a period of 5 years. We will use the data as part of another study (IF NEEDED).

Clinical examination (Specify details and purpose): NA

Blood sample collection: Specify quantity of blood being drawn: NA.

No. of times it will be collected: NA

Whether blood sample collection is part of routine procedure or for research (study) purpose:

1. Routine procedure 2. Research purpose

Specify **purpose**, discomfort likely to be felt and side effects, if any: NA

Whether blood sample collected will be stored after study period: NA

Whether blood sample collected will be sold: NA

Whether blood sample collected will be shared with persons from another institution: NA

Medication given, if any, duration, side effects, purpose, benefits: NA

Whether medication given is part of routine procedure: NA

Whether alternatives are available for medication given: NA

Final interview (specify approximate duration):_____NA____ mts. If **photograph** is taken, purpose: NA

Benefits from this study: FINDINGS LEARNT THROUGH THIS STUDY WILL AID IN BETTER TREATMENT & EARLY PREDICTION OF OUTCOME IN ST ELEVATION MYOCARDIAL INFARCTION USING ECG WHICH IS AN EASY & INEXPENSIVE TOOL.

Risks involved by participating in this study: NONE

How the **results** will be used: Study will be submitted to Dr. MGR medical university as thesis in post graduate course in general medicine.

If you are uncomfortable in answering any of our questions during the course of the interview / biological sample collection, **you have the right to withdraw from the interview / study at anytime.** You have the freedom to withdraw from the study at any point of time. Kindly be assured that your refusal to participate or withdrawal at any stage, if you so decide, will not result in any form of compromise or discrimination in the services offered nor would it attract any penalty. You will continue to have access to the regular services offered to a patient. You will **NOT** be paid any remuneration for the time you spend with us for this interview / study. The information provided by you will be kept in strict confidence. Under no circumstances shall we reveal the identity of the respondent or their families to anyone. The information that we collect shall be used for approved research purposes only. You will be informed about any significant new findings - including adverse events, if any, – whether directly related to you or to other participants of this study, developed during the course of this research which may relate to your willingness to continue participation.

Consent: The above information regarding the study, has been read by me/ read to me, and has been explained to me by the investigator/s. Having understood the same, I hereby give my consent to them to interview me. I am affixing my signature / left thumb impression to indicate my consent and willingness to participate in this study (i.e., willingly abide by the project requirements).

Signature / Left thumb impression of the Study Volunteer / Legal Representative:

Signature of the Interviewer with date:

Witness:

Contact number of PI: 9791524008

Contact number of Ethics Committee Office: 0422 2570170 Extn.: 5818

ஒப்புதல் படிவம்

தேதி :

மரு. சில்பித்தா .க, ஆகிய நான், பி. எஸ். ஜி. மருத்துவக் கல்லூரியின், பொது மருத்துவ துறையின் கீழ், **"மாரடைப்பு நோயினை கண்டறிய இருதய சுருள் படம் (இ.சி.ஜி) ஒரு முக்கிய அங்கம் வகிக்கிறது என்றும் ST அலைகள் ஏற்றம் ஏற்படும் மாரடைப்பால் இ.சி.ஜி யில் QRS அலைகளில் ஏற்படும் மாற்றங்கள் குறித்தும் மற்றும் மருத்துவ சிகிச்சையினால் அந்த நோய் எந்த அளவுக்கு குணமடையும் "** என்ற தலைப்பின் கீழ் ஆய்வு மேற்கொள்ள உள்ளேன்.

என் ஆய்வு வழிகாட்டி: மரு. எல். எஸ். சோமசுந்தரம்,

ஆய்வு மேற்கொள்வதற்கான அடிப்படை:

மாரடைப்பு நோயின் கூறுகளையும் பாதிப்புகளையும் ஏற்படாமல் தவிர்க்க அதற்கான மருத்துவ சிகிச்சைகளை மேற்கொள்வது.

ஆய்வின் நோக்கம்:

மாரடைப்பு நோய் உள்ளவர்களுக்கு இ. சி. ஜி மற்றும் எக்கோ (ECHO) ஸ்கேன் ஆகியவற்றில் ஏற்படும் மாற்றங்களுக்கு சம்மந்தம் இருப்பதை அறிந்து கொள்வதற்கு இந்த ஆய்வு மேற்கொள்ளப்படுகிறது.

ஆய்வு மேற்கொள்ளும் இடம்: பி. எஸ். ஜி. மருத்துவமனை, கோயம்புத்தூர்.

ஆய்வின் பலன்கள்:

மாரடைப்பு நோயின் கூறுகள் மற்றும் பாதிப்புகளை முன்னரே அறிந்து அதற்கான முறையான சிகிச்சையை மேற்கொண்டு நோயின் விளைவுகளையும் வீரியத்தையும் தவிர்க்கலாம்.

ஆய்வினால் ஏற்படும் அசௌகரியங்கள் / பக்க விளைவுகள்: பக்க விளைவுகள் எதுவும் இல்லை.

இந்த ஆய்வில் கிடைக்கும் தகவல்கள் 5 வருடங்கள் பாதுகாக்கப்படும். இவை தேவைப்பட்டால் வேறு ஆய்விற்கும் பயன்படுத்தப்படலாம். எந்த நிலையிலும் உங்களைப் பற்றிய தகவல்கள் யாருக்கும் தெரிவிக்கப்படமாட்டாது. அவை இரகசியமாக வைக்கப்படும்.

இந்த ஆய்வில் பங்கேற்க ஒப்புக்கொள்வதால் எந்த விதமான பலனும் உங்களுக்கு கிடைக்காது. எந்த நேரத்தில் வேண்டுமானாலும் ஆய்விலிருந்து விலகிக்கொள்ளும் உரிமை உங்களுக்கு உண்டு. ஆய்விலிருந்து விலகிக்கொள்வதால் உங்களுக்கு அளிக்கப்படும் சிகிச்சையில் எந்த வித மாற்றமும் இருக்காது.

இந்த ஆராய்ச்சிக்காக உங்களிடம் சில கேள்விகள் கேட்கப்படும் / சில இரத்த மாதிரிகள் அல்லது திசு மாதிரிகள் எடுக்கப்படும்.

மேலும், இந்த ஆய்வில் பங்கு கொள்வது உங்கள் சொந்த விருப்பம். இதில் எந்த விதக் கட்டாயமும் இல்லை. நீங்கள் விருப்பப் பட்டால், இந்த ஆய்வின் முடிவுகள் உங்களுக்குத் தெரியப் படுத்தப்படும்.

ஆய்வாளரின் கையொப்பம் :
தேதி :

ஆய்வுக்குட்படுபவரின் ஒப்புதல்:

நான் இந்த ஆராய்ச்சியின் நோக்கம் மற்றும் அதன் பயன்பாட்டினைப் பற்றி தெளிவாகவும், விளக்கமாகவும் தெரியப்படுத்தப் பட்டுள்ளேன். இந்த ஆராய்ச்சியில் பங்கு கொள்ளவும், இந்த ஆராய்ச்சியின் மருத்துவ ரீதியான குறிப்புகளை வரும் காலத்திலும் உபயோகப்படுத்திக் கொள்ளவும் முழு மனதுடன் சம்மதிக்கிறேன்.

ஆய்வுக்குட்படுபவரின் பெயர், முகவரி :

கையொப்பம் :
தேதி :

ஆய்வாளரின் தொலைபேசி எண்: 9791524008
மனித நெறிமுறைக் குழு அலுவலகத்தின் தொலைபேசி எண்: 0422 2570170 Extn.: 5818

LIST OF FIGURES

LIST OF TABLES

MASTER CHART

S.No	AGE	GENDER	DIABETES	HYPERTENSION	DYSLIPIDEMIA	SMOKER	ALCOHOLIC	AREA OF	RECIPROCAL CHANGES	QRS AMPLITUDE	LVD	WMS	INTERVENTION	OUTCOME AT FIRST FOLLOW
1	3	m	y	y	n	y	n	1	n	63	1	20	4	1
2	5	m	y	y	n	n	n	1	y	58	2	26	1	1
3	6	m	n	n	n	n	n	2	y	53.5	0	21	5	1
4	5	f	y	y	n	n	n	2	y	38	1	23	1	1
5	4	m	y	n	n	n	n	2	y	60	0	23	4	1
6	7	m	n	y	y	n	n	2	y	33	1	20	4	1
7	4	f	n	y	n	n	n	2	y	56.5	0	18	2	2
8	4	m	y	n	n	n	n	1	y	58	1	21	4	2
9	4	m	y	y	n	n	n	1	n	18	2	27	1	0
10	3	m	n	n	y	y	n	1	n	57	0	20	1	2
11	4	m	y	y	y	n	n	2	y	28	0	21	1	3
12	3	m	n	n	n	n	n	1	n	63	2	27	2	1
13	2	m	n	n	n	y	y	1	y	32	2	26	2	0
14	4	m	y	n	n	n	n	1	y	41.5	3	30	4	2
15	5	m	y	n	n	n	n	1	y	29.5	2	30	4	3
16	6	f	y	n	n	n	n	2	y	35	0	20	1	1
17	2	m	n	n	n	n	n	1	y	71	1	24	4	2
18	3	m	n	n	n	n	n	1	n	31	1	21	4	2
19	6	m	y	y	n	n	n	1	n	28	2	25	1	2
20	4	m	n	n	n	n	n	1	n	65.5	1	23	1	1
21	5	m	n	n	n	n	n	2	y	19	3	33	1	2
22	3	m	n	n	y	n	n	1	y	49	2	27	2	2
23	6	m	y	y	n	n	n	1	y	14	2	28	4	1
24	4	f	y	y	n	n	n	1	n	61	2	26	3	0
25		m	n	n	n	y	n	1	y	32	3	30	2	2
26	7	f	n	n	n	n	n	1	n	49.5	2	23	1	1
27	5	f	y	n	n	n	n	1	n	54	2	26	1	1
28	1	m	n	n	n	n	n	1	n	33	1	23	1	1
29	5	f	y	y	n	n	n	1	y	45.5	2	29	6	3
30	5	m	y	n	n	n	n	1	n	73	0	20	1	1

S.No	AGE	GENDER	DIABETES	HYPERTENSION	DYSLIPIDEMIA	SMOKER	ALCOHOLIC	AREA OF	RECIPROCAL CHANGES	QRS AMPLITUDE	LVD	WMS	INTERVENTION	OUTCOME AT FIRST FOLLOW
31	3	m	y	y	y	n	n	1	y	21	2	29	2	2
32	6	f	y	y	n	n	n	1	n	38.5	2	27	2	1
33	5	m	n	n	n	y	n	1	y	25.5	0	18	4	2
34	6	m	n	n	n	n	n	1	n	27.5	2	30	4	1
35	5	m	n	n	n	n	n	1	y	43.5	2	31	4	2
36	4	m	y	y	n	n	n	1	y	32	1	26	6	3
37	1	m	n	y	n	n	n	1	n	51	1	22	1	1
38	4	m	y	n	n	n	n	1	n	23.5	0	19	5	2
39	5	m	y	y	n	n	n	1	n	21	2	23	4	1
40	5	m	y	y	n	n	n	2	n	29	1	20	2	1
41	4	f	n	n	y	n	n	2	n	30	0	17	4	1
42	7	m	n	y	y	n	n	2	y	21	0	19	4	2
43	5	f	n	y	n	n	n	2	n	26	0	20	1	1
44	4	m	n	n	n	y	n	2	y	61	1	21	4	2
45	4	f	y	y	y	n	n	2	y	40.5	1	23	4	1
46	6	m	n	n	n	y	y	2	n	22	1	20	1	1
47	4	f	y	y	n	n	n	2	n	24	3	31	1	0
48	3	f	y	y	n	n	n	2	n	30	0	17	4	1
49	3	f	n	y	n	n	n	2	n	28	0	19	4	1
50	5	m	n	y	n	y	y	2	n	30	0	22	4	1
51	5	m	n	n	n	y	n	2	n	20	1	25	4	1
52	4	m	y	n	n	n	n	2	n	19	1	26	4	1
53	5	m	n	n	n	n	n	2	n	32	0	17	2	1
54	4	f	n	n	n	n	n	2	y	30	0	19	2	2
55	4	m	n	n	y	n	n	2	y	31	3	34	1	2
56	2	m	n	n	n	n	n	2	n	24	1	24	3	1
57	5	m	y	n	n	y	n	2	y	20	2	23	2	2
58	2	m	y	n	y	n	n	2	n	31	0	17	4	1
59	3	m	n	n	n	n	n	2	n	34	0	17	3	1
60	6	f	y	y	n	n	n	2	n	19	3	32	2	1
61	4	f	n	y	n	n	n	2	y	36	0	17	6	1

S.No	AGE	GENDER	DIABETES	HYPERTENSION	DYSLIPIDEMIA	SMOKER	ALCOHOLIC	AREA OF	RECIPROCAL CHANGES	QRS AMPLITUDE	LVD	WMS	INTERVENTION	OUTCOME AT FIRST FOLLOW
62	5	m	y	n	n	n	n	1	y	28	2	25	1	2
63	6	m	y	n	n	n	n	1	n	27.5	2	27	1	1
64	3	m	n	n	n	n	n	1	n	34	2	27	5	0
65	4	m	y	y	n	n	n	1	n	4	1	22	5	1
66	1	m	n	n	n	n	n	1	n	40	2	25	2	1
67	3	m	y	n	n	y	n	1	y	33	0	21	2	1
68	4	f	n	n	y	n	n	1	n	37	2	26	5	1
69	6	m	n	n	n	n	n	1	y	28	1	23	1	1
70	2	m	n	n	n	y	n	1	y	33.5	3	35	1	1
71	3	m	y	y	n	n	n	1	n	29	2	25	4	1
72	4	m	y	y	n	n	n	1	y	31.5	1	21	1	2
73	5	m	n	n	n	y	y	1	n	30	1	28	4	1
74	2	m	y	y	n	n	n	1	n	52	0	23	1	1
75	4	m	y	y	y	n	n	1	n	12	3	32	2	0
76	3	m	y	y	y	n	n	1	n	12	2	28	1	1
77	3	m	y	n	y	n	n	1	y	29	0	19	4	2
78	4	m	n	n	y	n	n	1	y	46	1	23	2	2
79	6	f	y	n	n	n	n	1	n	50	1	24	1	1
80	4	m	y	n	n	n	n	1	y	48	2	30	2	2
81	3	m	n	n	n	n	n	1	n	8.5	2	32	4	1
82	5	m	y	y	n	n	n	1	n	31.5	2	28	2	0
83	4	m	y	y	n	n	n	1	n	14	1	21	1	1
84	2	m	y	n	y	n	n	1	y	15	0	21	1	1
85	5	m	y	n	y	n	n	1	y	32	2	22	2	1
86	6	m	y	y	n	n	n	1	n	64	1	20	2	1
87	5	m	y	n	n	n	n	1	n	21	1	21	1	1
88	4	m	n	y	n	n	n	1	y	25	2	24	4	1
89	4	m	y	y	n	n	n	1	n	61	1	23	4	1
90	5	m	y	n	n	n	n	1	y	11	2	24	4	1
91	3	m	n	y	y	n	n	1	y	20	0	20	2	1
92	1	m	y	n	n	n	n	1	n	35	0	21	1	1

S.No	AGE	GENDER	DIABETES	HYPERTENSION	DYSLIPIDEMIA	SMOKER	ALCOHOLIC	AREA OF	RECIPROCAL CHANGES	QRS AMPLITUDE	LVD	WMS	INTERVENTION	OUTCOME AT FIRST FOLLOW
93	5	m	n	n	n	n	n	1	n	8	0	20	2	1
94	3	m	y	n	y	n	n	1	n	38	0	18	4	1
95	4	m	n	n	n	y	y	1	n	11	1	20	1	2
96	4	m	y	n	n	n	n	1	y	36	2	23	2	1
97	5	m	y	y	y	n	n	2	n	65	0	17	1	1
98	5	m	y	n	n	n	n	1	n	20	2	26	4	1
99	2	m	n	n	y	y	n	1	y	22	2	26	4	1
100	6	m	y	n	n	n	n	1	y	30	1	20	3	1
101	6	m	y	n	n	n	n	2	y	64	0	17	2	2
102	6	m	y	y	n	n	n	1	y	11.5	2	24	1	2
103	3	m	n	n	n	n	n	2	y	32	0	22	1	1
104	5	m	n	n	y	n	n	1	n	19	2	23	1	1
105	4	m	n	n	y	y	y	1	y	52	1	19	3	1
106	4	m	y	n	y	n	y	2	y	47.5	0	17	1	2
107	4	m	y	y	n	y	y	1	y	35	0	19	3	0
108	5	m	y	y	n	y	n	1	y	19	1	27	1	3
109	4	m	n	y	n	y	n	1	y	32	2	28	3	1
110	4	m	y	y	n	n	n	2	y	66	1	23	1	1
111	3	m	y	n	y	y	y	1	n	55.5	0	17	2	1
112	3	m	y	y	y	n	n	2	y	26	2	26	2	2
113	2	m	n	n	n	n	n	1	n	13	3	30	1	1
114	26	m	n	n	n	n	n	1	n	11	0	22	2	1

MASTER SHEET – KEY

AGE (YEARS)

18-29 →1

30-39 →2

40-49 →3

50-59 →4

60-69 →5

70-79 →6

>80 → 7

GENDER

Male – m

Female – f

DIABETES,HYPERTENSION,DYSLIPIDEMIA,

SMOKER,ALCOHOLISM

YES- y

NO - n

AREA OF INFARCT

ANTERIOR WALL – 1

INFERIOR WALL – 2

RECIPROCAL CHANGES

YES – y

NO – n

LVD

GOOD - 1

MILD - 2

MODERATE – 3

SEVERE – 4

WMS – WALL MOTION SCORE

INTERVENTION

MEDICAL – 1

THROMBOLYSIS – 2

PCI – 3

PTCA – 4

THROMBOLYSIS + PTCA – 5

CABG - 6

OUTCOME AT FIRST FOLLOW UP

LOST TO FOLLOW UP - 0

NYHA CLASS I – 1

NYHA CLASS II – 2

NYHA CLASS III - 3